12.3.3 Obesity

Overview

- Obesity affects nearly a third of adults in the U.S. and is associated with low-grade inflammation that potentially interact with PM-related inflammation.
- Evidence indicates the potential for dosimetric differences for $PM_{2.5}$ among adults and children by obesity status.
- Evidence from recent stratified epidemiologic analyses of long-term $PM_{2.5}$ exposure and mortality suggest increased risk for those who are obese compared to those who are not; evidence for other outcomes is inconsistent.
- Variability in the definition of obesity limits comparability between studies and the ability to distinguish risk between those who are overweight and obese.
- **Overall, the evidence is suggestive of increased risk for $PM_{2.5}$-related health effects among those who are obese compared to those who are not.**

In the U.S., obesity is defined as a BMI of 30 kg/m^2 or greater, with a BMI between 25 and 30 kg/m^2 indicating an overweight individual. It is a public health issue of growing importance as obesity rates in adults have continually increased over several decades in the U.S., reaching an estimated 30% in 2016 (Blackwell and Villarroel, 2018). Furthermore, 36% of adults in the U.S. are considered overweight while 34% are at a healthy weight (BMI 18.5−25 kg/m^2) (Blackwell and Villarroel, 2018). Obesity or high BMI could potentially increase the risk of PM related health effects through multiple mechanisms. For example, persistent low grade inflammation associated with obesity or excess nutrients and energy (CN and AR, 2011; Gregor and Hotamisligil, 2011; Lumeng and Saltiel, 2011) may work in conjunction with PM related inflammation that is thought to facilitate atherosclerotic plaque progression (Section 6.3.1, Figure 6-11). Obesity is closely related to diabetes, and is one component of metabolic syndrome, where co-occurring factors may also be associated with PM exposure (Section 7.2.1, Figure 7-2) and further facilitate cardiovascular risk (Section 6.3.1). Nutritional access and poor diet (Section 12.6.2) may also be potential risk factors that act in combination with obesity. Additionally, those who are obese may experience greater particle deposition in the lung as there is evidence of increased ventilation rates for overweight or obese adults and children, as well as a lower nasal breathing fraction and increase deposition fraction among obese children (Section 4.1.3, Section 4.2.4.4).

The 2009 PM ISA evaluated several studies that reported differences in subclinical cardiovascular and inflammatory markers between obese and nonobese participants in association with short-term exposure to $PM_{2.5}$(Dubowsky et al., 2006; Schwartz et al., 2005; Bennett and Zeman, 2004). A number of recent studies examining effect measure modification $PM_{2.5}$-related health effects by obesity statues are available and have reported some evidence of increased risk for mortality among obese individuals; however, evidence in studies across the range of effects examined including cardiovascular disease, incident diabetes, reproductive, and development outcomes do not consistently indicate differential risk by obesity status (Supplemental Table S12-3) (U.S. EPA, 2018).

Several studies examined effect measure modification of associations between mortality and long-term $PM_{2.5}$ exposure by obesity status. Overall, there was a trend across studies of increased risk among those who were overweight or obese compared to those of normal weight, though there are some exceptions to this trend across studies, and effect estimates are imprecise (i.e., wide 95% confidence intervals). A number of multicity studies in the U.S., Canada, and Europe reported increased risk for mortality among those who were obese (Villeneuve et al., 2015; Beelen et al., 2014a; Beelen et al., 2014b; Weichenthal et al., 2014; Puett et al., 2009). However, Turner et al. (2011) reported decreasing risk as BMI increased, including a 14% decrease in risk for those overweight compared to normal BMIs and a negative association among obese individuals. Furthermore, it is possible there is some variation by underlying cause of mortality. For example, Pinault et al. (2016) observed marginal decreases in risk for all-cause and cardiovascular mortality among those who were obese, though they reported a 35% increase in risk for respiratory mortality among obese participants. In contrast to these results, a pooled analysis of European cohorts observed that as BMI increased the association between $PM_{2.5}$ and respiratory mortality declined, while the opposite was true for all-cause and cardiovascular mortality (Beelen et al., 2014a; Beelen et al., 2014b; Dimakopoulou et al., 2014).

Studies have also examined a differential risk for a variety of cardiovascular effects by obesity status. In general, studies found little evidence for differences between obese and nonobese individuals, and when changes in association were present, they tended to be modest and imprecise. For example, a registry study of long-term $PM_{2.5}$ exposure and incident hypertension in Ontario, Canada (Chen et al., 2014) reported a decrease in risk for obese participants (HR: 1.07, 95% CI: 0.91, 1.26) compared to nonobese participants (HR: 1.17, 95% CI: 1.04, 1.33). Likewise, an examination of the Nurses' Health Study reported an increased risk in incident cardiovascular disease for obese participants (HR: 1.12, 95% CI: 0.99, 1.30) compared to nonobese participants (HR: 0.99, 95% CI: 0.88, 1.12) (Hart et al., 2015). A number of studies also examined changes in blood pressure with both long-term (Chan et al., 2015; Fuks et al., 2011) and short-term (Hoffmann et al., 2012; Wellenius et al., 2012b) exposures to $PM_{2.5}$ and observed no consistent pattern by obesity status for changes in blood pressure. Other studies examined outcomes such as prevalence of heart disease (Johnson and Parker, 2009) or measures of atherosclerosis (Hoffmann et al., 2009b) and did not observe an increase in risk among those who were obese compared to those with healthy weight.

Several of the studies that examined cardiovascular endpoints related to atherosclerosis and modification by diabetes status, as previously described (Section 12.3.2), also examined potential modification by obesity and observed limited evidence of increased risk among obese participants compared to those or healthy weight. In a study of the MESA cohort, Allen et al. (2009) identified positive $PM_{2.5}$ associations with elevated risk for calcification among obese individuals compared to those of normal weight. Furthermore, in those obese individuals with some or no calcification a positive change in the Agatston score (measure of coronary artery calcification) was observed. A similar study of the MESA cohort estimated the effect of 20 year $PM_{2.5}$ averages on atherosclerosis health measures and found no differences in association by BMI category (Roux et al., 2008). In a German population-based

cohort study (Heinz Nixdorf Recall study) Bauer et al. (2010) found a slightly stronger association between $PM_{2.5}$ exposure and carotid intima-media thickness (CIMT) for obese participants compared to those of normal weight.

Other studies specifically evaluated effect modification by obesity status on associations between markers of inflammation and coagulation, including IL-6, CRP, and fibrinogen. Hoffmann et al. (2009a) and Hertel et al. (2010) conducted analyses from German Heinz Nixdorf Recall Study cohort and found no distinct effect by obesity status on $PM_{2.5}$ associations with fibrinogen or CRP. A Study of Women's Health Across the Nation (SWAN), demonstrated increased CRP for middle aged obese women, though estimates had wide confidence intervals (Ostro et al., 2014).

A limited number of studies investigated effect measure modification by obesity for associations between $PM_{2.5}$ and other health endpoints, such as incident diabetes and reproductive outcomes. Among studies of incident diabetes, results were inconsistent. A study in Ontario, Canada reported decreased risk of developing diabetes among the overweight and obese (Chen et al., 2013), while multicity studies in Denmark (Hansen et al., 2016) and Germany (Weinmayr et al., 2015) reported increased risk among the obese compared to healthy weight. Among studies of reproductive outcomes, insufficient studies were available to report any trends for a specific outcome; however, there was little evidence of modification by obesity status in studies of endometriosis (Mahalingaiah et al., 2014), and gestational diabetes (Robledo et al., 2015). Conversely, in a small study of preeclampsia among predominantly Hispanic women in Los Angeles, Mobasher et al. (2013) reported higher risks among nonobese women based on $PM_{2.5}$ exposures in the first trimester compared to obese women.

Overall, the available evidence is suggestive of increased risk among those who are obese compared to those who are not obese for $PM_{2.5}$-associated health effects. There is a relatively consistent evidence across a small evidence base demonstrating increased risk of $PM_{2.5}$-associated mortality among those who are obese or overweight compared to those of healthy weight. Results from other outcomes were less consistent, although some studies observed increased risk in markers of atherosclerosis as well as incident diabetes. An important limitation across studies was the variability in categorizing obesity, with thresholds defining obesity ranging from a BMI of 27 to 30.6 kg/m^2. Furthermore, many studies did not distinguish between being overweight or obese and included overweight individuals either with obese individuals or with healthy weight individuals.

12.3.4 Elevated Cholesterol

Overview

- Elevated cholesterol is a common chronic condition in the U.S. adult population and is an important risk factor for other serious health conditions associated with $PM_{2.5}$ exposure, such as cardiovascular disease and diabetes.
- The 2009 PM ISA did not evaluate cholesterol status, but some recent studies have examined differences $PM_{2.5}$-associated health effects in the context of lipid disorders. This limited epidemiologic evidence provides evidence of increased risk with short- and long-term $PM_{2.5}$ exposure for those with elevated cholesterol compared to normal cholesterol.
- Additional epidemiologic studies stratifying by cholesterol medication (i.e., statins) usage provide limited evidence of increased risk of cardiovascular disease among statin users compared to those not taking statins.
- **Overall, the evidence is inadequate to determine if adults with elevated cholesterol are at increased risk for $PM_{2.5}$-related health effects.**

Elevated blood cholesterol is a common chronic health condition in the U.S., with the prevalence of hypercholesterolemia in the U.S. adult population approximately 26.0%, as reported by the 1999–2006 National Health and Nutrition Examination Surveys (Fryar et al., 2010). Metabolic disruption, such as dyslipidemia, can increase the risk of other health conditions, such as cardiovascular disease and diabetes. Additionally, as examined in Chapter 6 and Chapter 7, there is some evidence that short-term (Section 6.3.5, Section 7.1.3.3) and long-term (Section 6.3.12, Section 7.2.5.5) $PM_{2.5}$ exposures are associated with changes in blood lipids. While elevated blood cholesterol is an important health risk factor, few studies have explicitly investigated if blood cholesterol status increases the risk of other health outcomes associated with $PM_{2.5}$ exposure.

The PM 2009 ISA (U.S. EPA, 2009) did not evaluate studies examining potential differences in populations based on cholesterol. A limited number of epidemiologic studies have investigated differences between populations with and without high cholesterol, or by statin usage, and observed some evidence of higher risk for $PM_{2.5}$ related mortality and cardiovascular outcomes (Supplemental Table S12-4) (U.S. EPA, 2018). While these studies indicate those with elevated cholesterol, or those who use statins, may have potentially higher risks, overall, there were insufficient studies available to determine if cholesterol status consistently modifies health outcomes associated with $PM_{2.5}$ exposure.

In a study of 13 northeastern U.S. states, using data from the NHS cohort, Puett et al. (2009) evaluated the potential for effect measure modification by hypercholesterolemia status with $PM_{2.5}$ exposure over the 12-months prior to all-cause mortality, or a fatal coronary heart disease (CHD) event. In stratified analyses, the authors observed increased risk among those with hypercholesterolemia (HR: 1.53, 95% CI: 1.15–2.03) compared to those without hypercholesterolemia (HR: 1.04, 95% CI: 0.77–1.40). Puett et al. (2009) observed a similar trend among a smaller subset of fatal CHD cases. A

small study of myocardial infarction hospital admissions in Rochester, NY also observed a larger positive association among patients with history of dyslipidemia (Gardner et al., 2014).

In addition to studies with information on direct measures of blood cholesterol or patient history of dyslipidemia, several studies stratified study populations by use of statins or lipid-lowering medication. Long-term exposure studies in the U.S. and Germany (Bauer et al., 2010; Allen et al., 2009), as well as a meta-analysis of randomized controlled trials in Los Angeles (Künzli et al., 2010) observed increased risk of atherosclerosis associated with $PM_{2.5}$ exposure among those using statins compared to those not using statins. Studies of other health measures and long-term $PM_{2.5}$ exposure, such as history of peripheral vascular disease (Hoffmann et al., 2009b), and platelet counts (Viehmann et al., 2015) also observed increased risk among individuals using statins. A U.S. based study, using data from the MESA cohort, did not observe any substantial changes in $PM_{2.5}$-related flow-mediated dilation; however, they observed a positive association in baseline arterial diameter among those using statins compared to no change for those not using statins (Krishnan et al., 2012). Conversely, studies of short- and long-term exposure that investigated systemic inflammation found decreased responses for biomarkers of systemic inflammation among those using statins (Viehmann et al., 2015; Ostro et al., 2014; Hertel et al., 2010); however, many statins have anti-inflammatory properties complicating interpretation of these results.

Overall, the limited evidence is inadequate to determine if elevated cholesterol increases risk for $PM_{2.5}$-related health effects compared to cholesterol in the normal range. A single long-term exposure study reported elevated risk among those with hypercholesterolemia for $PM_{2.5}$-related mortality, while a single short-term study reported elevated risk of ST-Elevation Myocardial Infarction. Several studies examining biomarkers or preclinical measures of atherosclerosis and vascular function provide some evidence of elevated cardiovascular disease risk among statin users; however, the evidence base is small. Other studies examined if statin usage modified $PM_{2.5}$-related systemic inflammation; however, many statins have known anti-inflammatory properties, making these studies less informative in determining whether those with elevated cholesterol exhibited differential subclinical responses due to $PM_{2.5}$ exposure. Further limitations among studies of statins include the relatively low proportion of participants who used statins, leading to less precise estimates (i.e., wide 95% confidence intervals), as well as the difficulty in interpreting how representative statin prescription information is for control of blood lipid disorders among populations using statins.

12.3.5 Pre-existing Respiratory Disease

Overview

- The most common chronic respiratory diseases in the U.S. are asthma and COPD. Asthma affects a substantial fraction of the U.S. population, and it is the leading chronic disease among children. COPD primarily affects older adults and contributes to compromised respiratory function and underlying pulmonary inflammation.
- There is strong evidence indicating $PM_{2.5}$-associated respiratory effects among those with asthma, which forms the primary evidence base for the likely to be causal relationship between short-term exposures to $PM_{2.5}$ and respiratory health effects (Chapter 5).
- Few studies are available from the recent literature or in the 2009 PM ISA that inform whether those with asthma are at disproportionate risk for $PM_{2.5}$-related health effects compared to those without asthma.
- While there is some evidence of $PM_{2.5}$-related health effects in individuals with COPD, there are few studies from the current and previous ISAs with stratified analyses to compare effects in individuals with and without COPD
- **Overall, there is suggestive evidence that individuals with respiratory disease, particularly asthma, may be at increased risk for $PM_{2.5}$-related health effects compared to those without respiratory disease.**

Asthma

Approximately 8.3% of adults and 8.4% of children (age <18 years) in the U.S. currently have asthma (Blackwell and Villarroel, 2018), and it is the leading chronic illness affecting children. With regard to consideration of those with asthma potentially being at increased risk for a $PM_{2.5}$-related health effect, it is important to note that individuals with asthma, and children, tend to have a higher degree of oronasal breathing, which can result in greater penetration of PM into the lower respiratory tract (Section 4.1.3). Furthermore, there is limited evidence demonstrating that individuals with asthma may have altered clearance of particles (Section 4.3.4).

The 2009 PM ISA concluded that individuals with asthma may be more susceptible to health effects related to PM based on a limited number of epidemiologic studies for respiratory effects and controlled human exposure and animal toxicological studies demonstrating biological plausibility for asthma exacerbation with exposures to $PM_{2.5}$. Consistent with this, recent evidence evaluated in this ISA supports that there is likely to be a causal relationship between short-term exposure to $PM_{2.5}$ and respiratory effects, based primarily on evidence for asthma exacerbation in epidemiologic studies (Section 5.1.2) with supporting evidence across disciplines that provides biological plausibility (Section 5.1.1). Given this evidence, it is clear that individuals with asthma experience $PM_{2.5}$-related respiratory effects; however, evidence informing an increase in risk compared to those without asthma is limited.

There continue to be few studies that provide comparisons between individuals with and without asthma (Supplemental Table S12-5) (U.S. EPA, 2018). The 2009 PM ISA (U.S. EPA, 2009) included only a handful of epidemiologic and controlled human exposure studies examining $PM_{2.5}$ or CAPs exposures that provided some evidence for increased risk. Recent evidence is also limited to a few epidemiologic studies with stratified analyses for asthma for a variety of disparate outcomes. Of these studies, Watanabe et al. (2015) and Prieto-Parra et al. (2017) are most informative as they examined respiratory outcomes (i.e., lung function and symptoms) in children with and without asthma. Both studies demonstrated positive associations with short-term exposures to $PM_{2.5}$ for those without asthma, but symptoms and lung function decrements were of greater magnitude in children with asthma.

Other studies examined nonrespiratory outcomes. A study measuring cytokine responsiveness in blood samples collected from children with and without asthma in Germany demonstrated $PM_{2.5}$-related proinflammatory responses in children with asthma that were not observed in children without asthma for short-term exposures. In a multicity U.S. study in adults, $PM_{2.5}$ associated lung cancer mortality was greater in those with asthma compared to those without provide some evidence for increased risk in those with asthma compared to those without (Klümper et al., 2015; Turner et al., 2011). Bunch et al. (2011) conducted a study in Utah of hospital admissions with a primary diagnosis of atrial fibrillation and observed generally positive associations with $PM_{2.5}$ in those with and without asthma. In a study of diabetes incidence in Ontario, Canada, Chen et al. (2013) observed individuals with asthma to be at slightly decreased risk for diabetes with long-term exposures to $PM_{2.5}$ compared to those without.

Chronic Obstructive Pulmonary Disease (COPD)

Chronic lower respiratory disease, including COPD, was ranked as the third leading cause of death in the U.S. in 2011 (Hoyert and Xu, 2012). COPD comprises chronic bronchitis and emphysema and affects approximately 6.8 million adults in the U.S., respectively (Table 12-2). Given that people with COPD have compromised respiratory function and underlying systemic inflammation, it is plausible that they could be at increased risk for an array of $PM_{2.5}$-related health effects. Furthermore, there was some evidence to suggest differences in dosimetry, including greater deposition and impaired mucociliary clearance, that is also described in this ISA (Sections 4.2.4.7 and 4.3.4).

The 2009 PM ISA (U.S. EPA, 2009) described inconsistent results across a small evidence base examining differential $PM_{2.5}$-related respiratory effects in individuals with COPD and those without. In the current review, there continues to be limited evidence examining differential risk by COPD status and most of the available studies have focused on cardiovascular outcomes (Supplemental Table S12-5) (U.S. EPA, 2018). Wang et al. (2017) and Turner et al. (2011) observed greater risk for mortality associated with long-term exposures to $PM_{2.5}$ for those with COPD in a multicity study in the U.S. However, studies for cardiovascular hospitalizations (i.e., atrial fibrillation, myocardial infarction, acute coronary syndrome, and heart failure), incident hypertension, and diabetes incidence did not consistently demonstrate that those with COPD are at greater risk than those without in studies of short- and long-term

$PM_{2.5}$ exposures (Chen et al., 2014; Chen et al., 2013; Bunch et al., 2011; Belleudi et al., 2010; Rich et al., 2010; Haley et al., 2009). There are no recently published controlled human exposure studies that have examined health effects in individuals with COPD.

Despite limited evidence from epidemiologic and experimental studies examining $PM_{2.5}$-related health effects in those with and without pre-existing COPD, the evidence characterized in Chapter 5 demonstrates that there is evidence of COPD exacerbation associated with short-term exposure to $PM_{2.5}$ (Section 5.1.4), contributing to the conclusion of a "likely to be causal" relationship. In particular, epidemiologic studies report positive associations between $PM_{2.5}$ and hospital admissions and emergency department visits for COPD, with supporting evidence from panel studies demonstrating COPD exacerbation. Epidemiologic evidence is supported by limited experimental evidence of COPD-related effects, which provides biological plausibility for COPD in response to $PM_{2.5}$ exposure. This evidence indicates that $PM_{2.5}$-associated effects are observed in those with COPD, but it does not indicate if this risk is disproportionate compared to those without COPD.

Taken together, the collective evidence is suggestive that those with pre-existing respiratory diseases, particularly asthma and COPD, are at increased risk for $PM_{2.5}$-related health effects compared to those without pre-existing respiratory diseases. For asthma, there is strong evidence across disciplines indicating that there is likely to be a causal relationship for respiratory effects and $PM_{2.5}$ exposures based on asthma exacerbation, but few studies have conducted stratified analyses to inform increased risk. For COPD, the evidence base is limited to a few studies with inconsistent results for no respiratory outcomes.

12.4 Genetic Factors

Overview

- Variability in genetic background is known to contribute to the wide range of biological responses and diseases that are observed in the human population.
- Although limited, recent epidemiologic evidence is consistent with that characterized in the 2009 PM ISA, demonstrating differential risk for $PM_{2.5}$-related responses in individuals with variants in genes in the glutathione pathway that has a key role in oxidative stress.
- This is coherent with evidence supporting the biological plausibility for $PM_{2.5}$-related health effects as oxidative stress is an important early response following exposure.
- Several other genetic variants and epigenetic factors have been examined, but evidence is limited for each.
- **Overall, the evidence is suggestive that individuals with variants in the glutathione pathway are at increased risk for $PM_{2.5}$-related health effects compared to those without a variant genotype.**

Genetic variation in the human population is known to contribute to numerous diseases and differential physiologic responses. The potential for genetic background to modify responses to exposure to PM was evaluated in the 2009 PM ISA (U.S. EPA, 2009) and the biological plausibility of individuals with certain genotypes known to result in reduced function in genes encoding antioxidant enzymes being at increased risk for respiratory effects related to ambient air pollution was described. Though the evidence base for any particular genetic polymorphism was limited, the 2009 ISA concluded that evidence suggested that specific genetic polymorphisms could potentially increase the susceptibility of an individual to health effects related to PM exposure. In the recently published literature, several additional studies are available that examine genes related to antioxidant defense, inflammation, and lipid metabolism (Supplemental Table S12-6) (U.S. EPA, 2018).

Glutathione is the primary antioxidant defense in the body and is critical to protecting against oxidative stress. Because of this, variant genotypes in the glutathione pathway have been the most commonly studied with regard to health effects related to PM because oxidative stress is known to be one of the early biological responses following exposure (Sections 5.1.2, 5.2.1, 6.1.1, and 6.2.1). The 2009 PM ISA described results from a few studies that observed those with GSTM1 null genotypes to be at increased risk for cardiovascular effects related to $PM_{2.5}$ exposures (Schneider et al., 2008; Chahine et al., 2007; Schwartz et al., 2005). Of the recent epidemiologic evidence examining genetic variants in the oxidative stress pathway, only one study provides additional evidence for cardiovascular outcomes. Hampel et al. (2010) demonstrated that in adults with prior MI, $PM_{2.5}$-related QTc prolongation was greater in individuals with the minor allele for NFE2L2 rs1364725 compared to those with the major allele. Other recent evidence examines respiratory outcomes in children and provides additional support for effect measure modification by genetic background. For example, in a study of elementary and middle school children in Taiwan, $PM_{2.5}$-related increases in leukocytes and neutrophils in nasal lavage samples were greater in those with GSTM1 null genotypes compared to GSTM1 positive (Chen et al., 2016). Another study examined haplotypes in the glutathione synthetase gene (GSS) utilizing data from the Children's Health Study in southern California. While stratification for GSS haplotype 010000 demonstrated larger decrements in FVC in association with long-term $PM_{2.5}$ concentrations compared to other haplotypes, slightly smaller decrements in FEV1 and MMEF were observed for haplotype 010000 (Breton et al., 2011). Fuertes et al. (2013) conducted a pooled-analysis of 6 birth cohorts across Europe to examine associations in doctor-diagnosed allergic rhinitis at 7−8 years of age among variants for SNPs in GSTP1, TNF, TLR2, and TLR4. This study found positive associations for $PM_{2.5}$ and allergic rhinitis across all children, and magnitude of association in children with the minor alleles for GSTP1 (rs1138272) was slightly larger than those homozygous for the major allele. In addition, the magnitude of association between $PM_{2.5}$ and allergic rhinitis was slightly larger for those having minor alleles for SNPs in TNF (rs1800629) and TLR4 (rs10759930), implicating an inflammatory response with long-term exposure to $PM_{2.5}$.

Other studies examined a diverse range of genetic variants and outcomes. Wilker et al. (2011) examined modification of the $PM_{2.5}$-associated changes in adhesion molecules by genetic variants in

micro-RNA processing genes in the participants from the Normative Aging Study. Relatively little is known about the role of these genes relative to inflammation, but this study demonstrated that those having the minor allele for GEMIN4 (rs1062923) had lower levels of ICAM-1 and VCAM-1 in association with short-term $PM_{2.5}$ exposures. Ren et al. (2010) also used data from the NAS to evaluate genetic background, though the focus of this study $PM_{2.5}$-related HRV and modification by polymorphisms in lipid metabolism and endothelial function. A number of polymorphisms were examined in APOE, LPL, and VEGF and results demonstrated that the minor allele for the SNPs examined was associated with smaller reductions in HRV. Lastly, Hampel et al. (2012) examined effect modification by SNPs associated with cardiovascular outcomes as identified in the literature and demonstrated inconsistent results for CHT1 rs333229, rs2966762, rs1871841 and $PM_{2.5}$-related decrements in HRV, though the relevance of these SNPs is not clear.

Some recently published animal studies have also examined genetic variants, particularly in relation to PM-induced metabolic effects. Experimental genetic knockout studies in mice exposed to $PM_{2.5}$ support a role for TLR4 activation of Nox2 leading to a systemic inflammation (Kampfrath et al., 2011). In another study of mice deficient in the CC-chemokine receptor 2 (CCR2) gene, defective monocyte recruitment during immune responses were protected from $PM_{2.5}$ and high fat diet induction of hepatic steatosis, insulin resistance, systemic and peripheral inflammation (Liu et al., 2014). Other studies utilized a mouse model deficient in the neutrophil NADPH oxidase gene (required for superoxide anion production) and found that they were protected from CAPs-induced increases in superoxide production, insulin resistance, increase in abdominal mass and visceral adiposity, and fibrosis in mice (Zheng et al., 2015; Xu et al., 2010).

Recent evidence has also included the examination of DNA methylation and the underlying role it may play in $PM_{2.5}$-related health effects. Across the studies of DNA methylation (Peng et al., 2016; Lepeule et al., 2014; Bind et al., 2012; Salam et al., 2012), hypermethylation of a number of genes have been examined including iNOS, ICAM1, CRAT, ICAM, IFN-gamma, IL-6, iNOS, OGG1, GCR, F3, and TLR2. While there is some evidence that hypermethylation of these genes may play a role in mediating $PM_{2.5}$-related health effects when compared to hypomethylation, evidence is too limited to draw conclusions.

Overall, the evidence is suggestive that individuals with genetic variants in the glutathione pathway are at increased risk for $PM_{2.5}$-related health effects compared to those without variant genotypes. There is consistent evidence from a handful of studies in the recent literature and the 2009 PM ISA demonstrating that variants in the glutathione pathway may increase the risk of a PM-related health effect that is supported by evidence for biological plausibility and a role for oxidative stress in initial responses to exposures to $PM_{2.5}$. A variety of other variants have been examined in addition to studies of DNA methylation in $PM_{2.5}$-related health effects, but the evidence is too limited to determine if they modify risk.

12.5 Sociodemographic Factors

12.5.1 Lifestage

The 2009 ISA for Particulate Matter (U.S. EPA, 2009) discussed some evidence for increased risk of health effects related to PM exposure among different lifestages (i.e., children and older adults). Lifestage refers to a distinguishable time frame in an individual's life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth (U.S. EPA, 2014). Differential health effects of PM across lifestages could be due to several factors. With regard to children, the human respiratory system is not fully developed until 18−20 years of age, and therefore, it is biologically plausible that children may have intrinsic risk for respiratory effects due to potential perturbations in normal lung development. Older adults, typically considered those 65 years of age or greater, have weakened immune function, impaired healing, decrements in pulmonary and cardiovascular function, and greater prevalence of chronic disease (Table 12-2), which may contribute to, or worsen health effects, related to PM exposure. Also, exposure or internal dose of PM may differ across lifestages due to varying ventilation rates, increased oronasal breathing at rest, and time-activity patterns. The following sections present the evidence comparing lifestages from the recent literature, which builds on the evidence presented in the 2009 Particulate Matter ISA (U.S. EPA, 2009).

12.5.1.1 Children

Overview

- Children makeup a substantial fraction of the population and often have unique risks because of their continuous growth and development.
- Limited recent evidence indicates that children may have higher $PM_{2.5}$ exposures than adults and that there are dosimetric differences in children compared to adults.
- Strong evidence demonstrates $PM_{2.5}$-associated health effects in children, particularly from recent epidemiologic studies of short-term $PM_{2.5}$ exposure and impaired lung function growth, decrements in lung function, and asthma development.
- Evidence from stratified analyses in the current and previous ISAs demonstrates generally positive associations with $PM_{2.5}$ exposure of similar magnitudes for children and adults.
- **Overall, evidence is adequate that children are at increased risk for $PM_{2.5}$-related health effects, with the strongest evidence from associations with effects specifically examined in children (e.g., lung function growth and asthma development).**

Children may be particularly at risk for health effects related to ambient $PM_{2.5}$ exposures compared to adults due to (1) children's developing respiratory system, (2) children's increased ventilation rates relative to body mass compared to adults, and (3) the increased proportion of oral

breathing observed among children, particularly boys, relative to adults. Such oral breathing can result in higher exposures compared to nasal breathing (Section 4.2.4.2). In addition, children tend to spend more time outdoors, and, consequently, have the potential for greater exposure to ambient $PM_{2.5}$. Consistent with these opportunities for greater exposure, Bell and Ebisu (2012) observed higher $PM_{2.5}$ exposures among children and young adults (0–19 years) compared to adults (20–64 years). According to the 2010 census, 24% of the U.S. population is less than 18 years of age, with 6.5% less than age 6 (Howden and Meyer, 2011). The large proportion of children within the U.S. supports the public health significance of characterizing the risk of PM-related health effects among children.

While there is some evidence to inform dosimetric and exposure differences among children (Sections 4.2.4 and 4.3.4), there has been little evidence from stratified analyses to demonstrate children being at increased risk of the health effects associated with $PM_{2.5}$ exposure compared to adults. That is, positive effect estimates are often observed in stratified analyses of children, but these effect estimates are similar in magnitude to those observed for adults (Supplemental Table S12-7) (U.S. EPA, 2018). For example, recent studies of short-term $PM_{2.5}$ exposure and respiratory hospital admissions or ED visits report consistent, positive associations among analyses restricted to children; the magnitude of these associations is similar to those observed for adults (Atkinson et al., 2016; Samoli et al., 2016; Xu et al., 2016). Overall, the evidence from recent studies is consistent with previously evaluated evidence. The 2004 PM AQCD, summarizing studies examining either PM_{10} or $PM_{2.5}$, concluded that the "rather small group of studies does not show striking differences in effect estimates from analyses across age group strata" (U.S. EPA, 2004). The 2009 PM ISA(U.S. EPA, 2009) presented evidence from a single study of $PM_{2.5}$ (Mar et al., 2004) that observed stronger respiratory effects in children (7–12 years) compared to adults (20–51 years).

Other epidemiologic studies did not stratify results by lifestage, but instead restricted the analyses to children, and provide evidence for the occurrence of effects for a particular lifestage (i.e., effects that can only be observed in children). This is the case for a number of longitudinal studies of long-term $PM_{2.5}$ exposure and lung development (Section 5.2.2.1.1), lung function (Section 5.2.2.2.1), and asthma development (Section 5.2.3.1) in children. Recent longitudinal studies, particularly those from the Children's Health Study (CHS), are consistent with and extend the evidence that was available in the 2009 PM ISA demonstrating that long-term $PM_{2.5}$ exposure is associated with impaired lung function growth, decrements in lung function, and increased incidence of asthma development in children. Toxicological studies provide support for these associations in children as pre- and post-natal exposure to ambient levels of urban particles were found to impair mouse lung development. Recent results from the CHS not only corroborate previous results, but they also indicate improvements in lung development in association with declining $PM_{2.5}$ concentrations (Gauderman et al., 2015). In addition, a number of recent prospective and retrospective cohort studies based in North America and Europe provide evidence that long-term $PM_{2.5}$ exposure is associated with asthma development in children (Section 5.2.3.1).

Additional studies compared different age groups within the childhood lifestage. (Ding et al., 2016) evaluated asthma ED visits in Chongqing, China and observed higher effect estimates among 2−5 year old children compared to 0−1 or 6−18 year old children, though the inability to reliably diagnose asthma in younger children may contribute to the heterogeneity in these results. When considering ED visits due to pneumonia in Jinan, China, (Lv et al., 2016) reported higher effect estimates for infants (<1 year old) and young children (1−4 years old) compared to older children (5−15 years old).

In summary, the evidence demonstrating $PM_{2.5}$-associated health effects in children is adequate to conclude that children are at increased risk for $PM_{2.5}$-related health effects. There is strong evidence that children are at increased risk to the effects of $PM_{2.5}$ exposure, based primarily on studies examining effects specific to children. Epidemiologic studies of long-term $PM_{2.5}$ exposure demonstrate associations with impaired lung function growth (Section 5.2.2.1.1), decrements in lung function (Section 5.2.2.2.1), and increased incidence of asthma development in children (Section 5.2.3.1). The evidence from stratified analyses provides limited evidence that children are at increased risk of $PM_{2.5}$-related health effects compared to adults. In addition, there is some evidence indicating that children receive higher $PM_{2.5}$ exposures than adults and there are dosimetric differences in children compared to adults that can contribute to higher doses. Finally, there is emerging evidence from two Chinese studies suggesting that ages 1 to 5 years could be a critical window among children during which they experience respiratory health effects associated with short-term $PM_{2.5}$ exposure.

12.5.1.2 Older Adults

Overview

- Older adults represent an increasing portion of the U.S. population and often have pre-existing diseases/conditions that may compromise biological function.
- Limited recent evidence does not indicate that older adults have higher $PM_{2.5}$ exposures than younger adults, though older adults could receive higher doses due to dosimetric differences.
- Consistent evidence demonstrates $PM_{2.5}$-associated health effects in older adults, particularly between short- and long-term $PM_{2.5}$ exposure and mortality as well as cardiovascular or respiratory morbidity.
- Evidence from stratified analyses in the current and previous ISAs demonstrates similar associations with $PM_{2.5}$ exposure in older adults and younger adults.
- Animal toxicological and controlled human exposure studies provide additional evidence for the occurrence of effects among this particular lifestage, but do not inform whether or not this lifestage is at increased risk to the health effects of $PM_{2.5}$.
- **Overall, while $PM_{2.5}$-associated effects are observed in older adults, evidence is inadequate to determine if older adults are at increased risk for effects compared to younger adults.**

Older adults are a potentially at increased risk population due to the higher prevalence of pre-existing cardiovascular and respiratory diseases found in this age range compared to younger life stages. The increased risk in this lifestage can likely be attributed to the gradual decline in physiological processes that occurs with aging (U.S. EPA, 2006). Therefore, some overlap exists between populations considered to be at-risk due to pre-existing disease and lifestage (i.e., older adults) (Kan et al., 2008). According to the 2014 National Population Projections issued by the U.S. Census Bureau, approximately 14.9% of the U.S. population is age 65 years or older, and by 2040, this fraction is estimated to grow to 21.7% (U.S. Census Bureau, 2014); accessed November 9, 2017. Thus, this lifestage represents a substantial proportion of the U.S. population demonstrating the public health importance of characterizing the potential for increased risk for health effects related to $PM_{2.5}$ exposure in this age group.

The 2009 ISA for Particulate Matter (U.S. EPA, 2009) indicated that compared with younger adults, older adults (typically ages 65 years and older) may be susceptible to PM-related cardiovascular effects. The evidence from epidemiologic, controlled human exposure and animal toxicological studies were generally consistent and coherent in supporting this conclusion, though some geographic heterogeneity in the pattern of associations among studies conducted in U.S. and non-U.S. locations was acknowledged. Additional evidence for associations between short-term PM exposure and respiratory morbidity and mortality was also available, and generally limited to results from epidemiologic studies.

Recent studies contribute to the existing body of evidence evaluating whether: (1) older adults experience higher exposures to $PM_{2.5}$ compared to younger adults; (2) stratified analyses conducted in epidemiologic studies support increased risk of health effects among older adults compared to younger adults; (3) animal toxicological, controlled human exposure, and epidemiologic analyses restricted to older populations provide coherence for the occurrence of effects for this particular lifestage, and (4) there is evidence for variability in associations among different age groups within the older adults lifestage.

Clearance of $PM_{2.5}$ from all regions of the respiratory tract decreases with increasing age beyond young adulthood in both humans and laboratory animals, indicating that older adults could receive higher doses of $PM_{2.5}$ compared to younger adults (Section 4.3.4). However, there is little evidence indicating that older adults are systemically exposed to higher concentrations of $PM_{2.5}$ than other lifestages. Miranda et al. (2011) observed that older adults (i.e., 65+ years) were less likely to live in counties with the highest daily or annual $PM_{2.5}$ concentrations. Consistent with this, Bell and Ebisu (2012) observed similar $PM_{2.5}$ exposures among older adults (65+ years) compared to adults (20−64 years).

A relatively large number of recent epidemiologic studies of short- and long-term $PM_{2.5}$ exposure and cardiovascular and respiratory health effects, as well as mortality, report generally consistent, positive associations among analyses restricted to older adults, though the magnitude of these associations is similar to those observed for younger adults (Supplemental Figure S12-8) (U.S. EPA, 2018). Studies of short-term $PM_{2.5}$ exposure and cardiovascular or respiratory effects generally consist of evaluations of hospital admission, emergency department visits, or mortality conducted in the U.S., Canada, Europe, or China. Generally, positive associations were observed for both younger adults and older adults with no

indication that the associations observed for older adults were consistently greater in magnitude. A number of studies of long-term $PM_{2.5}$ exposure evaluated associations with cardiovascular effects among older adults and younger adults and did not observe stronger magnitude of effects among the older adults. Evaluations of subclinical cardiovascular effects (e.g., blood pressure, measures of vascular functions, concentrations of circulating biomarkers) were somewhat less consistent in demonstrating positive associations with long-term $PM_{2.5}$ concentrations compared to cardiovascular mortality. Similar to the results of studies of long-term $PM_{2.5}$ exposure and cardiovascular mortality, both short- and long-term $PM_{2.5}$ exposures were consistently associated with total (nonaccidental) mortality, but there was no indication that these associations were of greater magnitude in older adults compared to younger adults (Supplemental Figure S12-8) (U.S. EPA, 2018).

Though there are a relatively large number of epidemiologic studies evaluating the associations between $PM_{2.5}$ concentrations and health effects as detailed in (Supplemental Figure S12-8), it is noteworthy that there is substantial variability in the age ranges included as the reference group. For example, sometimes the reference group included all individuals less than a certain age (e.g., 60, 65, or 70 years), while other times the reference group included individuals from a smaller, more restricted range of ages (e.g., 35–64, 40–69, or 45–64 years). Such variability in the reference groups makes it difficult to make comparisons about the magnitude of effects across studies, though it should not affect inferences about whether older adults are at increased risk of $PM_{2.5}$-related health effects compared to younger adults. Additionally, it is possible that the results of stratified analyses could be affected by publication bias; several studies conducted stratified analyses by lifestage but did not report quantitative results when no differences were observed across strata. While likely to exist, such publication bias is unlikely to influence any inferences drawn from the body of evidence evaluated here, as these studies also did not generally observe differences in associations across age strata. Finally, some studies compared associations for older adults to those for all ages (including the older adults). Since these are not truly stratified analyses, and there is overlap between the two groups, results from those studies are not considered here nor included in Supplemental Figure S12-8 (U.S. EPA, 2018)

Several animal toxicological, controlled human exposure, and epidemiologic studies did not stratify results by lifestage, but instead restricted the analyses to older individuals, and can provide coherence and biological plausibility for the occurrence of effects among this particular lifestage. When considering animal toxicological studies, the 2009 PM ISA reported that exposure to $PM_{2.5}$ CAPs was associated with arrhythmias in older, but not younger rats. Recent studies extend the evidence that was available in the 2009 PM ISA from controlled human exposure studies demonstrating that $PM_{2.5}$ CAPs exposure is associated with decreases in HRV in older, healthy adults. In Copenhagen, Denmark, Hemmingsen et al. (2015) exposed older overweight, but healthy men and women to traffic-related air pollution (TRAP) that was nonfiltered or particle filtered and observed decreased high frequency measurements and increased low frequency measurements when nonfiltered TRAP was compared to particle filtered. In a dietary intervention study, Tong et al. (2012) reported that after a 28-day

supplementation period with olive oil, there was a lower HF/LF ratio immediately after CAP exposure in older adults. There were no changes in HRV time domain measurements found in this study.

Recent epidemiologic panel studies have observed associations with cardiovascular morbidity and $PM_{2.5}$ exposure among older adults (Sections 6.2.2.2, 6.2.6.2, and 6.2.11.1). In one study of older adults with ischemic heart disease in nursing homes in Los Angeles, CA, $PM_{2.5}$ concentrations were associated with ST-segment depression (Delfino et al., 2011). In addition, panel studies of older adult populations report generally consistent evidence for an association between short-term $PM_{2.5}$ exposure and BP, particularly studies including participants living in nursing homes or senior communities which allow for improved exposure assessment (Jacobs et al., 2012; Wellenius et al., 2012b; Liu et al., 2009). Among studies of inflammatory markers, the evidence was less consistent. Some panel studies of older adults observed positive associations between $PM_{2.5}$ and inflammatory IL6 and TNF in a (Wittkopp et al., 2013; Delfino et al., 2009), while others did not (Wang et al., 2016a; Rich et al., 2012; Liu et al., 2009).

Additional studies compared different age groups within the older adult lifestage. For example, Bell et al. (2015) observed higher magnitude effect estimates among those 85+ years compared to those aged 65–74 or 75–84 years for cardiovascular mortality, but not for respiratory mortality and short-term $PM_{2.5}$ exposure. Conversely, Madsen et al. (2012) observed higher effects among those aged 65–74 compared to those aged 74–85 or 85+ when examining short-term $PM_{2.5}$ exposure and total mortality. When evaluating long-term $PM_{2.5}$ exposure and total mortality and cardiovascular mortality, Crouse et al. (2015) observed positive associations for both men and women across age groups (i.e., 60–69, 70–79, 80–89 years). This is inconsistent with evidence reported in the 2009 PM ISA, where limited evidence indicated declines in effect estimates for mortality with increasing age, starting at 60 until there was generally a null association among individuals 85+ years. Overall, there is no consistent evidence that risk varies for different age groups within the older adult lifestage.

Overall, there continues to be evidence supporting that $PM_{2.5}$-associated health effects are present in older adults; however, the evidence is inadequate to determine whether older adults are at increased risk of $PM_{2.5}$-related health effects when compared to younger adults. Among epidemiologic studies of short- and long-term $PM_{2.5}$ exposure, there is little evidence to support increased risk of health effects among older adults compared to younger adults. While there is limited evidence that changes in physiology could result in decreased ability to clear $PM_{2.5}$ from the respiratory tract, there is no evidence that older adults are exposed to high $PM_{2.5}$ concentrations than younger adults. Animal toxicological, controlled human exposure, and epidemiologic studies continue to support that older adults are at risk to the effects of $PM_{2.5}$ exposure, especially cardiovascular effects. This evidence comes mainly from epidemiologic panel studies of short-term $PM_{2.5}$ exposure observing associations with cardiovascular morbidity among older adults residing in nursing homes, decreases in HRV in controlled human exposure studies of older adults, and increased arrhythmias in older rats in animal toxicological studies. Studies that did not stratify results by lifestage, but instead restricted the analyses to older individuals, provide coherence and biological plausibility for the occurrence of effects among this

particular lifestage. Finally, there is no consistent evidence to indicate that any age groups within the older adult lifestage have higher risks than others.

12.5.2 Sex

Overview

- Males and females in the U.S. have differing health concerns; for example, health effects related to reproduction (e.g., sperm motility in males and pregnancy outcomes in females) are sex-specific.
- For health outcomes concerning both sexes, there is some evidence of higher mortality in males than in females from long-term exposures to $PM_{2.5}$.
- For other health outcomes from long-term $PM_{2.5}$ exposure, and for outcomes from short-term $PM_{2.5}$ exposure, there is no clear pattern of increased risk for either sex.
- **Overall, the evidence in inadequate to determine if males are at increased risk for $PM_{2.5}$-related health effects compared to females.**

A large number of health conditions resulting in morbidity and mortality have been shown to differ by sex. The Centers for Disease Control and Prevention estimate that a male born in the U.S. in 2012 has a life expectancy of 76.4 years, while a female has a life expectancy of 81.2 years (Arias et al., 2016). Due to both biological and social differences it is reasonable to consider that the risks of exposure to air pollution may differ between sexes. For example, exposure risks related to gestation and fetal development will primarily concern females and differences between sexes in time spent at the workplace or at home (U.S. BLS, 2017) will potentially contribute to differences in PM exposure. Sex-specific biological risks related to fertility are described in Chapter 9 of this document. Briefly, health outcomes specifically concerning males include potentially decreased sperm motility (Radwan et al., 2015; Hammoud et al., 2009). Outcomes specifically concerning females involve pregnancy-related morbidity; this includes outcomes such as gestational hypertension, preterm birth, and low birth weight. Overall, evidence in Chapter 9 was considered suggestive of a causal relationship between $PM_{2.5}$ exposure and these sex-specific reproductive health concerns.

The 2009 PM ISA (U.S. EPA, 2009) concluded that neither sex had a consistently stronger association between PM exposure and health effects. Evidence from the recent literature generally supports this conclusion, though there may be specific outcomes that differ in risk by sex. Due to the lower life expectancy of males in the U.S., females have been selected as the "reference" category; however, either sex could be considered a potential "at-increased-risk" group of interest.

There is some evidence for differences in mortality due to PM exposure by sex, with males having potentially stronger associations than females (Supplemental Table S12-9) (U.S. EPA, 2018). Di et al. (2017) analyzed long-term $PM_{2.5}$ exposure and mortality in the U.S. Medicare population and found

a higher association for males (RR: 1.087, 95% CI: 1.083, 1.090) than for females (RR: 1.060, 95% CI: 1.057, 1.063). However, this was not the case for Medicaid-eligible (low-income) Medicare recipients, who did not display this difference between the sexes. While this is among the more comprehensive studies on this topic, other results of national U.S.-based long-term exposure studies have been inconsistent. A study by Wang et al. (2017) which includes an overlapping study population with that of Di et al. (2017) focuses on Medicare beneficiaries in the Southeastern U.S. only, and consistent with Di et al. (2017), the mortality-PM association within this region was also stronger for males than for females. Other studies report results ranging from males having roughly the same risk (Thurston et al., 2015) to slightly lower risk (Zeger et al., 2008) than females. In Canada, Crouse et al. (2015) reported higher PM-associated mortality among males in each age bracket considered and higher mortality among males as a group overall. The short-term $PM_{2.5}$-related effect differences on mortality by sex are negligible, with Huang et al. (2012) and Madsen et al. (2012) reporting slight increases for all-cause mortality in males and Samoli et al. (2013) reporting a slight decrease for males for non-accidental mortality.

Other studies have examined effect measure modification by sex for $PM_{2.5}$-associated cardiovascular effects. In a study of hospitalizations for U.S. Medicare beneficiaries, Bell et al. (2015) reported higher risks for females than for males from short-term $PM_{2.5}$ exposure for cardiovascular outcomes overall, as well as for heart rhythm disturbance and heart failure specifically. However, this observation was found to vary geographically, and this disparity was more pronounced in the Northeast than in other regions of the U.S. (Bell et al., 2015). In contrast, a study of short-term $PM_{2.5}$ exposure in Little Rock, Arkansas demonstrated that males had a greater association than females for cardiovascular-related emergency room visits (Rodopoulou et al., 2015). Short-term exposure studies conducted outside the U.S. have reported associations larger in magnitude for cardiovascular mortality in females (Milojevic et al., 2014) and congenital heart disease in males (Ye et al., 2016). However, in general for short-term exposure to $PM_{2.5}$, there is little evidence supporting the presence of disparities in cardiovascular outcomes between males and females. Specifically, in studies examining cardiovascular outcomes overall (Lanzinger et al., 2016; Kloog et al., 2014), cardiovascular mortality (Su et al., 2015), cardiac arrest (Silverman et al., 2010), heart failure (Haley et al., 2009), hypertension (Brook and Kousha, 2015), infarctions (Weichenthal et al., 2016; Rich et al., 2010), pulmonary embolism (Dales et al., 2010), and venous thrombosis (Dales et al., 2010) there was little difference in the magnitude of associations between males and females.

Similarly, evidence does not indicate disparities in cardiovascular outcomes from long-term $PM_{2.5}$ exposure. As with short-term exposures, disparities may vary by the specific characteristics of the population. A study of the U.S. population as a whole found little difference in CVD mortality by sex (Thurston et al., 2015), yet a study focused on families in the agricultural sectors of Iowa and North Carolina found somewhat higher mortality risk in males (Weichenthal et al., 2014). In general, however, recent long-term $PM_{2.5}$ exposure studies show only minor differences in outcomes by sex for heart disease (Wong et al., 2015; Johnson and Parker, 2009), hypertension (Chen et al., 2014; Johnson and Parker,

2009), blood pressure (Fuks et al., 2011), and cardiovascular disease or cardio-metabolic disease in general (Crouse et al., 2015; Wong et al., 2015).

There is little evidence for disparities in respiratory outcomes between males and females from long-term $PM_{2.5}$ exposures. A study of 50–71 year-olds in the U.S. found only a minor increase in respiratory mortality for women compared to men (Thurston et al., 2015). Conversely, a meta-analysis of European studies found a minor increase in men compared to women (Dimakopoulou et al., 2014). Wong et al. (2015) found little evidence of effect modification by sex for respiratory outcomes in Hong Kong. For short-term $PM_{2.5}$ exposure, Bell et al. (2015) found somewhat increased association in females for respiratory hospital admissions overall as well as for respiratory tract infections specifically. Other studies have found only negligible differences between males and females for respiratory hospital admissions (Lanzinger et al., 2016; Liu et al., 2016; Rodopoulou et al., 2015), pediatric asthma (Gleason et al., 2014), and peak expiratory flow (Watanabe et al., 2015).

Overall, the evidence is inadequate to determine if males are at increased risk for $PM_{2.5}$-associated health effects compared to females. There is some evidence that males may have higher mortality risk due to long-term $PM_{2.5}$ exposure than females. However, for other health outcomes associated with long-term $PM_{2.5}$ exposure as well as for morbidities resulting from short-term $PM_{2.5}$ exposure, there is inconsistent evidence that either males or females are at higher risk. In considering this evidence, it is also important to note that certain health outcomes are sex-specific. For example, there is some evidence for effects related to gestation that apply only to females and are not represented in sex-stratified studies, but this evidence is also inconsistent.

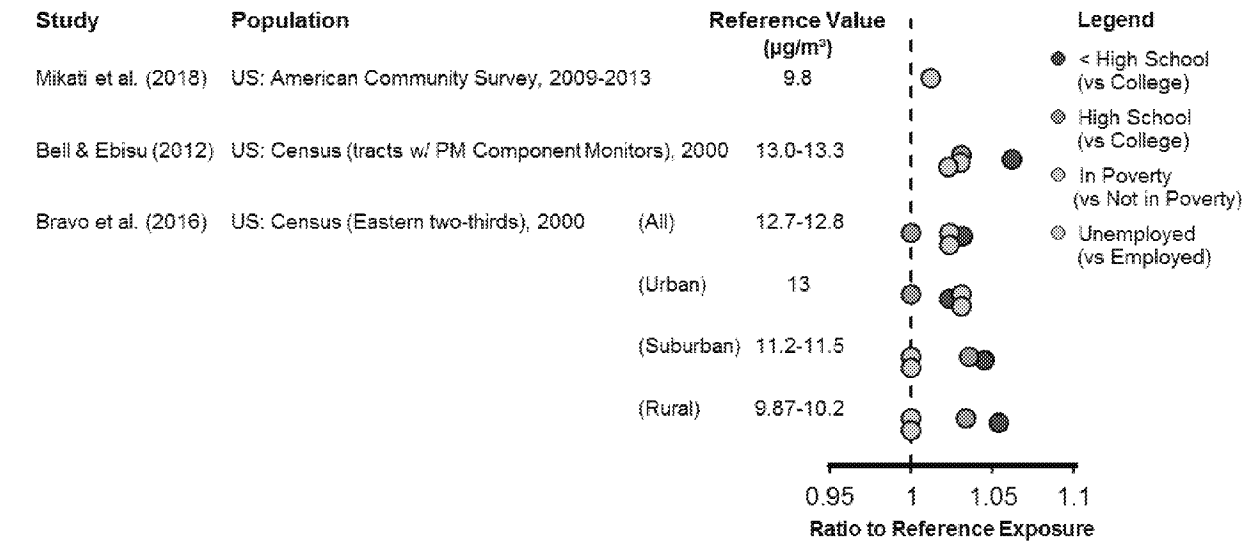
12.5.3 Socioeconomic Status

Overview

- Socioeconomic status (SES)—a composite measure that can include metrics such as income, education, or occupation—plays a role in access to healthy environments as well as access to healthcare in the U.S. Thus, SES may underlie differential risk for $PM_{2.5}$-related health effects.
- There is some evidence that demonstrates that having low income or living in lower-income areas results in stronger associations between mortality and long-term $PM_{2.5}$ exposures compared to higher-income counterparts.
- There is no clear pattern of differential risk when comparing effects in those with low educational attainment compared to higher educational attainment.
- **Taken together, the combination of exposure disparities and health evidence is suggestive that low SES populations are at increased risk for $PM_{2.5}$-related health effects compared to higher SES populations.**

Socioeconomic status (SES) is a composite measure that can represent various interrelated factors including income, education, or occupation—both in terms of the individual and in terms of the surrounding population's composition. The variety of metrics that fall under the umbrella of SES makes it difficult to make direct comparisons; for example, an income that is considered low in a particular city may be higher on the distribution of income at the national level. Furthermore, differences in social conditions from country to country make comparisons with studies taking place outside the U.S. difficult. However, it is still important to consider differential risk for $PM_{2.5}$-related health effects for SES. According to the U.S. Census Bureau, 12.7% of the U.S. population are living in poverty as of 2016 (Semega et al., 2017); 10.9% of the population aged 25 years and older does not have a high school diploma (U.S. Census Bureau, 2017a). Lower SES can impact place of residence and thus exposure to pollutants; it may be correlated with pre-existing health conditions that are potentially aggravated by air pollution; and it may result in inequities in access to resources such as healthcare.

Disparity in exposure to $PM_{2.5}$ due to differences in ambient $PM_{2.5}$ at the place of residence is one way in which SES may be related to PM risk (Figure 12-1). Mikati et al. (2018) compared modeled ambient $PM_{2.5}$ data for census tract populations across the U.S. and reported exposure to slightly higher concentrations of $PM_{2.5}$ for those living below the poverty line. Bell and Ebisu (2012) reported that those with less than a high school education, the unemployed, and those below the poverty line are exposed to higher concentrations of $PM_{2.5}$ (and to several $PM_{2.5}$ components) than do their higher-SES counterparts. Bravo et al. (2016) reported that lower educational attainment (no college degree) was associated with exposure to high $PM_{2.5}$ concentrations in suburban and rural areas (as well as urban areas when limiting to those without a high school diploma), and poverty status and unemployment were associated with exposure to high $PM_{2.5}$ concentrations in urban areas.



Note: Group for reference exposure listed in parentheses under legend.
Source: Permission pending, Mikati et al. (2018), Bell and Ebisu (2012), Bravo et al. (2016).

Figure 12-1 Differences in $PM_{2.5}$ exposure by socioeconomic status (SES).

The 2009 PM ISA (U.S. EPA, 2009) found some evidence for increased risk of mortality due to short-term $PM_{2.5}$ exposure in low-SES individuals. More recent studies have added to our understanding of the relationship between SES and PM-related health effects., including evidence where a variety of SES metrics and categories have been simplified into "high," "medium," and "low" status.

Several studies examined differential risk for $PM_{2.5}$-related mortality by SES level (Supplemental Table S12-11) (U.S. EPA, 2018). An expansive study examining the association between long-term exposure to $PM_{2.5}$ and mortality in the cohort of all Medicare beneficiaries in the U.S. reported that low-SES individuals, as measured by Medicaid eligibility, had a higher risk of $PM_{2.5}$-related mortality than high-SES individuals (Di et al., 2017). Another pair of studies focusing on the Medicare population reported that those living in low-income neighborhoods or low-SES cities have a slightly higher risk of long-term $PM_{2.5}$-related mortality than those in higher-income neighborhoods or higher-SES cities (Wang et al., 2017; Kioumourtzoglou et al., 2016). Residents of low-SES ZIP codes have slightly higher risk of mortality from long-term PM exposure than residents of high-SES ZIP codes in the Eastern, Central, and Western U.S. (Zeger et al., 2008). Studies conducted in Canada have reported similar results (Crouse et al., 2015; Brook et al., 2013). Mortality outcomes from a study of short-term $PM_{2.5}$ exposure in Norway reported slightly decreased risk in low-SES areas compared to higher-SES areas (Madsen et al., 2012).

Studies focusing on educational attainment have reported mixed results. Lee et al. (2015) reported that the risk of mortality from short-term $PM_{2.5}$ for those in their study area of GA, NC, and SC was more than doubled in the group that had eight or fewer years of education compared to the group having more

than eight years of education. While at least one European study reported lower risk of $PM_{2.5}$-related mortality for low-education individuals (Beelen et al., 2014a), other studies in the U.S. have reported either negligible differences by education status (Thurston et al., 2015) or higher risk of $PM_{2.5}$-related mortality for lower-education individuals (Kloog et al., 2013).

There is little evidence that the effect of $PM_{2.5}$ exposure on cardiovascular health outcomes is modified by SES. Coogan et al. (2016) conducted an analysis focused on long-term $PM_{2.5}$ exposure in a cohort of black women; among this subset of the population, risk of hypertension as a result of $PM_{2.5}$ was somewhat more pronounced in women outside the highest quintile of neighborhood SES, raising the possibility that race and SES interact. Kloog et al. (2014) reported that the increase in hospital admissions from short-term $PM_{2.5}$ exposure was greater in low income groups than in high income groups; however, other studies reporting CVD effects for both short-term (Haley et al., 2009) and long-term exposure (Johnson and Parker, 2009) have not reported this to be the case. A German study on the effects of long-term $PM_{2.5}$ exposure on blood pressure found no increase in risk for the unemployed compared to the employed (Fuks et al., 2011).

Results of CVD studies using education attainment as a metric of SES have been inconsistent (Supplemental Table S12-10) (U.S. EPA, 2018). Thurston et al. (2015) reported little difference in long-term $PM_{2.5}$-related CVD mortality between those with less than a high school education and those with greater than a high school education. Those with exactly a high school level education, however, had somewhat higher associations than either of these two groups. Increased CVD risk within an intermediate educational group was also reported by Coogan et al. (2016) which showed that participants with some college education had higher risk of hypertension from long-term $PM_{2.5}$ exposure than did college graduates or those without any college education. Johnson and Parker (2009) reported slightly higher associations for heart disease and for hypertension from long-term $PM_{2.5}$ exposure in lower-education individuals. Studies outside the U.S. have not shown that lower education individuals are more at risk for cardiovascular outcomes (Chen et al., 2014; Fuks et al., 2011).

The evidence that SES modifies the association between respiratory morbidity from PM exposure is also weak. Multiple Atlanta-based studies examining short-term $PM_{2.5}$ exposure and asthma reported results including slightly higher odds of asthma attacks for those in high-poverty ZIP codes and for those who were eligible for Medicaid, as well as those with lower maternal educational attainment (O'Lenick et al., 2017; Strickland et al., 2014; Sarnat et al., 2013). Another study based in New Jersey found little distinction in outcomes between low, moderate, and high-SES participants (Gleason et al., 2014). Thurston et al. (2015) reported that long-term exposure and respiratory mortality were not more strongly associated for lower-education groups than for those with more than a high school education.

Taken together, the combination of exposure disparities and health evidence is suggestive that low SES populations are at increased risk for $PM_{2.5}$-related health effects compared to populations of higher SES. Several studies show increased risk of overall $PM_{2.5}$-related mortality for

lower-income groups, but the metrics for income vary widely across studies. In addition, there is also weak evidence for differential risk for $PM_{2.5}$-related outcomes by educational attainment.

12.5.4 Race

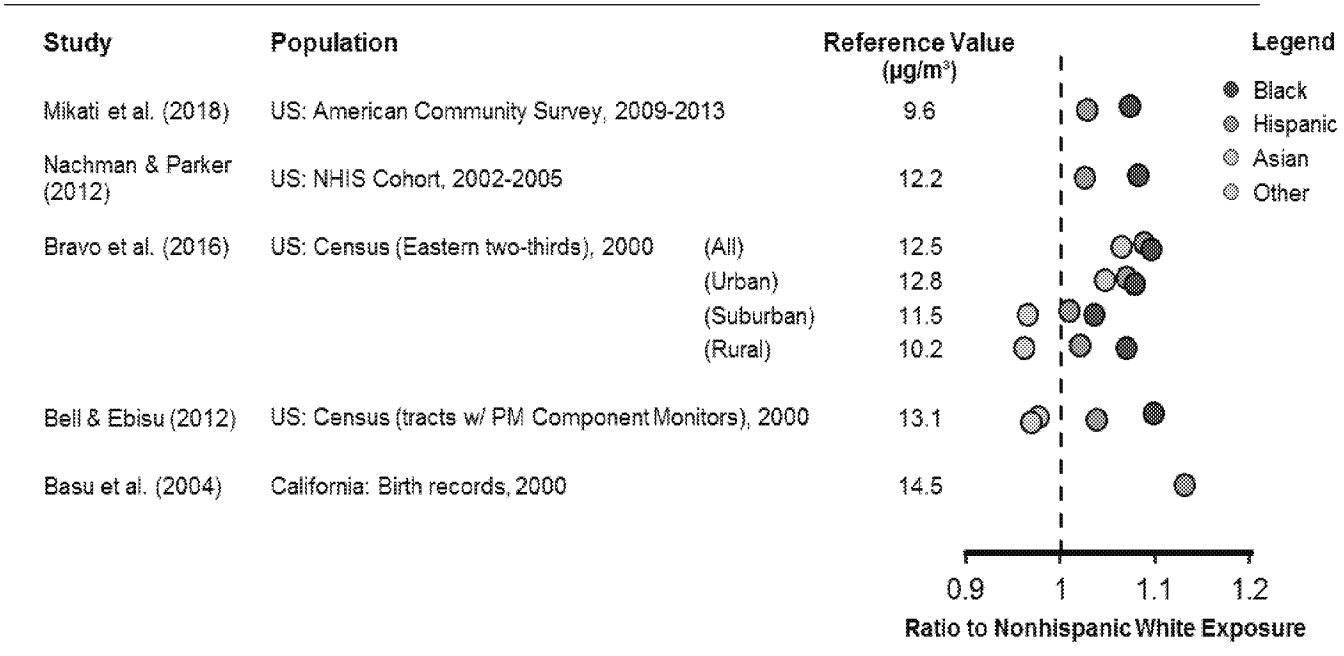
Overview

- People of different racial and ethnic backgrounds often have different health status disparities. The 2009 PM ISA found little evidence for increased $PM_{2.5}$-related risk by race and some evidence of increased risk by Hispanic ethnicity.
- Recent evidence demonstrates that there are consistent racial and ethnic disparities in $PM_{2.5}$ exposure across the U.S., particularly for blacks and African Americans compared to Nonhispanic whites.
- Recent studies provide evidence consistent with increased $PM_{2.5}$-related mortality from long-term exposure in blacks/African Americans; for $PM_{2.5}$-related health effects besides mortality there is also a general pattern of racial and ethnic disparities.
- **Overall, there is adequate evidence that race and ethnicity modify $PM_{2.5}$-related risk and that nonwhites, particularly Blacks, are at increased risk for $PM_{2.5}$-related health effects, in part due to disparities in exposure.**

Race and ethnicity are not biological categories but instead represent social definitions that broadly correspond to national origins (U.S. Census Bureau, 2017b). The U.S. Census Bureau considers racial categorization (e.g., white; black or African American; Hispanic; American Indian or Alaskan Native; Asian; Native Hawaiian or other Pacific Islander) to be distinct from ethnic categorization (e.g., Hispanic origin), but studies often examine race and ethnicity as a single concept (U.S. Census Bureau, 2017a). Furthermore, studies conducted outside of the U.S. may differ in the cultural and historical backgrounds that define race and ethnicity. Because of the fluidity of these categorizations, direct comparisons of results stratified by race and ethnicity between studies can be difficult. The evaluation of evidence for race and/or ethnicity in this section is done according to classifications made by original study authors.

The 2009 PM ISA (U.S. EPA, 2009) found little evidence that race and some evidence that ethnicity might be effect measure modifiers of PM-related mortality. However, this conclusion did not include an assessment of whether there is evidence of racial and ethnic disparities in PM exposure. Disparities in exposure to PM are one potential cause of disparity in PM-related health effects by race and ethnicity. Mikati et al. (2018) compared modeled ambient $PM_{2.5}$ data with census tract populations across the U.S. and reported higher exposures for Hispanics (9.9 $\mu g/m^3$) and higher exposures for Nonhispanic blacks (10.3 $\mu g/m^3$) than for Nonhispanic whites (9.6 $\mu g/m^3$). Nachman and Parker (2012) found that blacks in the nationally-representative 2002−2005 National Health Interview Survey were exposed to higher concentrations of ambient $PM_{2.5}$ (13.2 $\mu g/m^3$) than were Hispanics (12.5 $\mu g/m^3$) or Nonhispanic

whites (12.2 μg/m^3). Hispanics in this sample had only slightly higher exposures than Nonhispanic whites, but in some specific areas, the disparities may be larger. For example, a study of year 2000 birth records in the state of California reported a higher mean $PM_{2.5}$ concentration at monitors within five miles of Hispanic residences (18.2 μg/m^3) compared to Nonhispanic white (15.8 μg/m^3) residences over the gestation period (Basu et al., 2004). Disparities appear to persist in urban, suburban, and rural environments (Bravo et al., 2016). Hispanics and Blacks as well as Asians are also exposed to higher concentrations of certain components of $PM_{2.5}$ (such as elemental and organic carbon) than are Nonhispanic whites (Bell and Ebisu, 2012). In addition, Johnson and Parker (2009) reported that more blacks and Hispanics lived in high-exposure (≥15.8 μg/m^3) census block groups than whites.



Note: Group for reference exposure is Nonhispanic Whites.

Source: Permission pending, Mikati et al. (2018), Nachman and Parker (2012), Bravo et al. (2016), Bell and Ebisu (2012), Basu et al. (2004).

Figure 12-2 Differences in $PM_{2.5}$ exposure by race.

A further limitation to the discussion of race in the 2009 PM ISA (U.S. EPA, 2009) was the small number of studies available at the time. For instance, evidence for modification of short-term $PM_{2.5}$ mortality risk by Hispanic ethnicity primarily came from two studies in California: Ostro et al. (2006) and Ostro et al. (2008). However, a number of studies published since the 2009 PM ISA have considered effect measure modification by race and ethnicity.

A number of epidemiologic studies that examined the association between long-term $PM_{2.5}$ exposure and mortality reported that race/ethnicity modifies this relationship (Supplemental Table S12-12) (U.S. EPA, 2018). There is evidence for elevated risk among Nonwhites compared to Whites. Kioumourtzoglou et al. (2016), (Wang et al., 2017), and (Arnaud, 2011) all examined long-term $PM_{2.5}$-related mortality in the U.S. Medicare population and found racial disparities in in mortality risk. Kioumourtzoglou et al. (2016) found higher long-term $PM_{2.5}$-related mortality among residents of cities at the 75th percentile of proportional black population than among those in cities at the 25th percentile. Di et al. (2017) observed that whites had a lower risk for long-term $PM_{2.5}$-related mortality (RR: 1.063; 95% CI: 1.060, 1.065) than the overall population while Hispanics (RR: 1.116; 95% CI: 1.100, 1.133) and Asians (RR: 1.096; 95% CI: 1.075, 1.117) had higher risk; blacks, meanwhile, had greater risk (RR: 1.208; 95% CI: 1.199, 1.217) than either of these groups. Furthermore, the researchers showed that this discrepancy was not explained by low economic status alone; blacks with a high enough income to be ineligible for Medicaid retained greater risk than Medicaid-eligible whites. However, within a 1997–2009 National Health Interview Survey cohort, Parker et al. (2017) did not find significant differences by race or ethnicity in all-cause or heart disease mortality. Wang et al. (2017), which focused on the Medicare population only in the Southeastern U.S., found a greater mortality risk from long-term $PM_{2.5}$ exposure for blacks than for whites in this region. A study focused only on mortality records in the states of Georgia, North Carolina, and South Carolina reported a greater increase in short-term $PM_{2.5}$-associated mortality among the black population as well (Lee et al., 2015).

Beyond studies of mortality, other recently published literature has examined whether there is evidence of effect measure modification by race/ethnicity on the relationship between long-term $PM_{2.5}$ exposure and cardiovascular effects. Johnson and Parker (2009) reported that only Hispanics had a significantly elevated risk for heart disease associated with long-term $PM_{2.5}$ exposure; only whites had a significantly elevated risk for hypertension.

Studies focused on smaller geographic areas have reported inconsistent results. Among the 2000–2002 Multiethnic Study of Atherosclerosis cohort recruited from six cities across the U.S., Hicken et al. (2016) observed a larger mean difference in left-ventricular mass index (an outcome related to hypertension) associated with long-term $PM_{2.5}$ exposure in Blacks as opposed to Whites. However, they did not report such a difference between groups for left-ventricular ejection fraction (another outcome related to hypertension). Similarly, a study of over 80,000 cases of cardiovascular-related ED visits in Central Arkansas did not find a significant racial difference in outcomes for short-term $PM_{2.5}$ exposures (Rodopoulou et al., 2015); nor did a study of transmural myocardial infarctions in New Jersey (Rich et al., 2010).

In addition, there is evidence that associations between $PM_{2.5}$ exposures and respiratory outcomes are stronger for nonwhites than whites. Nachman and Parker (2012) observed that asthma prevalence associated with long-term $PM_{2.5}$ exposure was statistically significantly higher in Nonhispanic blacks, but not in Hispanics, than in Nonhispanic. There is also some evidence of effect measure modification by race

for short-term $PM_{2.5}$ exposures and respiratory effects. Short-term $PM_{2.5}$-related respiratory risks focused on individual cities are inconsistent. Glad et al. (2012) observed a slight increase in odds of asthma ED visits for African Americans compared to whites associated with short-term $PM_{2.5}$ exposure in Allegheny County, PA from 2002–2005. Alhanti et al. (2016), on the other hand, investigated asthma-related ED visits in Atlanta, GA, Dallas, TX, and St. Louis, MO between 1993–2009 and did not observe pronounced differences between whites and nonwhites in associations with $PM_{2.5}$ either overall or within any specific age ranges or individual cities.

Strickland et al. (2014) focused on pediatric asthma in Atlanta from 2002–2010 and the relationship with a population-weighted city average for short-term $PM_{2.5}$ from several monitors. They observed higher risk associated with $PM_{2.5}$ on pediatric asthma ED visits for African Americans compared to non-African Americans, and this difference was more prominent than differences based on other measures such as education or Medicaid status. Gleason et al. (2014) focused on pediatric asthma ED visits in a 2005–2007 New Jersey cohort and did not find any significant difference in outcomes by black or white race, but did observe a significantly increased odds ratio of events in those of Hispanic ethnicity as opposed to Nonhispanic ethnicity. The Central Arkansas study by Rodopoulou et al. (2015) reported lower short-term $PM_{2.5}$-related risk of respiratory emergency room visits for African Americans.

While evidence for reproductive effects is only suggestive of, but not sufficient to infer, a causal relationship with exposure to PM2.5 (Chapter 9), a limited number of studies evaluated whether race/ethnicity modified the relationship between $PM_{2.5}$ exposure and reproductive outcomes, including adverse birth outcomes and maternal effects during pregnancy; they provide mixed evidence for greater risk among nonwhites. Bell et al. (2007) conducted a study of births in Massachusetts and Connecticut between 1999–2002, assigning $PM_{2.5}$ as the average of all monitors in a county. They noted a larger decrease in birthweight for black mothers than they did for white mothers. Pereira et al. (2014) overlapped with the time and geography of Bell et al. (2007) by considering preterm birth in Connecticut from 2000–2006. $PM_{2.5}$-related preterm birth was lower for children of white mothers (OR: 1.02; 95% CI: 0.88, 1.20) than for children of black mothers (OR: 1.39; 95% CI: 0.99, 1.96) or Hispanic mothers (OR: 1.31; 95% CI: 1.00, 1.73). Among Hispanic mothers, odds of preterm birth were uniquely high for $PM_{2.5}$ exposure within the first trimester (OR: 1.25; 95% CI: 1.08, 1.44). Green et al. (2015) modeled zip code-level $PM_{2.5}$ exposure in California from 1999–2009 and compared to over 5.5 million birth records in the state. They did not find differential effects for stillbirth by race or ethnicity. Vinikoor-Imler et al. (2012) analyzed the risk of gestational hypertension associated with $PM_{2.5}$ exposure in North Carolina between 2000–2003. They reported a significantly lower risk of gestational hypertension for Hispanics than for whites, but a significantly higher risk for blacks.

Overall, there is adequate evidence that nonwhites, particularly blacks, are at increased risk for $PM_{2.5}$-related health effects based on studies examining differential exposure and health effects. There is strong evidence demonstrating that black and Hispanic populations, in particular, have higher $PM_{2.5}$ exposures than Nonhispanic white populations. In addition, there is consistent evidence across

multiple studies demonstrating an increase in risk for Nonwhite populations. More specifically, effect measure modification by race in high-quality studies of $PM_{2.5}$-associated mortality (Di et al., 2017; Wang et al., 2017) are complemented by studies examining effect modification on $PM_{2.5}$-associated morbidity.

12.5.5 Residential Location

Overview

- New methods in exposure assessment allow for the estimation of $PM_{2.5}$ exposures for both urban and rural populations, evidence indicates that $PM_{2.5}$ is generally lower in rural areas compared to urban areas.
- Studies examining exposure differences in populations with close proximity to roadways indicate $PM_{2.5}$ concentrations are generally not elevated close to roadways.
- Evidence is inconsistent across stratified epidemiologic analyses examining health effects compared to degree of urbanicity (e.g., urban or rural residence).
- There is some evidence from epidemiologic and toxicological studies that demonstrates an increase in risk for those exposed to traffic particles or live near a roadway.
- With fewer available $PM_{2.5}$ monitor sites in smaller metropolitan and rural locations compared to larger metropolitan areas, the ability to validate modeled ambient $PM_{2.5}$ in less populated locations remains an important limitation; furthermore, the diversity in residential classification metrics limits the ability to interpret trends across studies.
- **Overall, the evidence is inadequate to determine if residential location increases risk for $PM_{2.5}$-related health effects.**

12.5.5.1 Urban/Rural Residential Locations

Many studies examining the health effects of $PM_{2.5}$ exposure have traditionally focused on urban populations due to the predominantly urban siting of monitors in the national monitoring network; however, those living outside major metropolitan areas may be exposed to different mixtures of particulate matter than those in urban areas (Xu et al., 2015) and this may vary across regions in the U.S. (Sections 2.5.3 and 3.4.4.1). Residential location may also be an important surrogate for other factors, including differing access to services, lifestyle, and other environmental exposures that could potentially influence $PM_{2.5}$-health associations (Grabich et al., 2016). Recent developments in estimating exposure through hybrid models drawing from satellite observations, chemical transport model output, and ambient concentration measurements to estimate ambient $PM_{2.5}$ concentrations have enabled a greater proportion of rural populations to be included in recent epidemiologic studies (Section. 3.3.2). These studies have not only examined whether overall associations between $PM_{2.5}$ and health outcomes are present with the addition of rural populations, but have also examined differences in associations between urban and rural populations. These new methods also provide the opportunity to examine if there are differences in associations by degree of urban density or urbanicity, as many previous studies relied on a limited number of fixed site monitors in large metropolitan areas.

Few studies in the 2009 PM ISA reviewed the potential for modification by residential location, and those that did often incorporated residential information only as a general surrogate for socioeconomic status. However, a study in Phoenix did note that the largest association between mortality and short-term $PM_{2.5}$ exposure was in an area of medium urban density in central Phoenix (Wilson et al., 2007). Recent studies have examined whether degree of urbanicity modifies the association between $PM_{2.5}$ exposure and a variety of health effects. These studies report inconsistent results with the majority of studies focusing on mortality and long-term $PM_{2.5}$ exposure.

$PM_{2.5}$ concentrations are generally lower in rural areas compared to urban areas in the U.S., based both on limited monitoring data, as well as remote-sensing and hybrid modeled $PM_{2.5}$ estimates (Section 2.5.3). Several epidemiologic studies reported average $PM_{2.5}$ stratified by varying definitions of urban and rural residential location and generally observed similar trends of lower $PM_{2.5}$ in rural areas. Average annual rural $PM_{2.5}$ in the U.S. ranged from 10.2−12.9 $\mu g/m^3$, while urban $PM_{2.5}$ ranged from 11.5−15.5 $\mu g/m^3$ (Bravo et al., 2017; Garcia et al., 2015; Strickland et al., 2015). Moreover, there are compositional characteristics of urban ambient $PM_{2.5}$ that are consistent with traffic emissions and have been shown to change when moving away from the urban center (Section 2.5.1.2.5).

There is some evidence of stronger associations between $PM_{2.5}$ exposure and mortality in urban areas compared to rural areas; however, evidence is inconsistent across various metrics that use different categorization schemes based on the population size of a city or city urbanicity (Supplemental Table S12-13) (U.S. EPA, 2018). Di et al. (2017) and Kioumourtzoglou et al. (2016) both examined long-term $PM_{2.5}$ exposure and mortality using nationwide Medicare data, though the latter focused on the variation of urbanicity, rather than a comparison to nonmetropolitan areas.. Using modeled $PM_{2.5}$ across the entire continental U.S., as well as the largest Medicare study population to date, Di et al. (2017) observed stronger positive associations between $PM_{2.5}$ exposure and mortality in areas of moderate population density compared to areas of high population density. Meanwhile, Di et al. (2017) observed a smaller positive association among areas of low population density. Kioumourtzoglou et al. (2016) observed no strong evidence of modification in a pooled analysis of 207 U.S. cities by degree of urbanicity or population density within cities. However, in region specific metaregression the authors observed that as population density and urbanicity increased, there were larger effects for $PM_{2.5}$-mortality in the Northeast, Midwest, and Northwest compared to the South, Southeast, Central, Southwest, and Western regions of the U.S.

Additional multicity studies in the U.S, including six Northeastern states (Shi et al., 2015), seven Southeastern states (Wang et al., 2017), and Massachusetts (Kloog et al., 2013) also used hybrid models to estimate long-term $PM_{2.5}$ exposure and observed some evidence of decreased risk of mortality in rural populations compared to urban populations. This difference in effect also persisted in models simultaneously stratified by race and sex (Wang et al., 2017). Conversely, a study of diabetes-related mortality and long-term $PM_{2.5}$ exposure in Canada observed a larger, but imprecise (i.e., wide 95% confidence intervals), association in rural areas compared to large or mid-population cities (Brook et al.,

2013). A study in California also observed higher rates of cardiovascular, cardiopulmonary, and overall mortality in rural compared to urban zip codes, though the strength of this pattern varied substantially by $PM_{2.5}$ exposure assignment method (Garcia et al., 2015). In addition to studies of long-term $PM_{2.5}$ exposure, a study of mortality and short-term $PM_{2.5}$ exposure in Georgia, North Carolina, and South Carolina observed higher risks in rural zip codes compared to metropolitan urban cores (Lee et al., 2015).

A limited number of studies evaluated if urbanicity characteristics modified the association between other health effects and $PM_{2.5}$, such as cardiovascular effects, respiratory effects and reproductive outcomes. In a study of long-term $PM_{2.5}$ exposure, Johnson and Parker (2009) observed attenuated associations in less-urban areas for self-reported cardiovascular disease, but larger associations for self-reported hypertension in urban areas.

Among a limited number of studies for short-term $PM_{2.5}$ exposure, studies of cardiovascular effects were inconsistent, while studies of respiratory effects tended to see increasing risk in less urban areas. Kloog et al. (2014) observed a negative association in urban areas, and no association in rural areas using Medicare data on cardiovascular hospital admissions. Conversely, using Medicare data in 708 U.S. counties, Bravo et al. (2017) reported increasing associations between short-term $PM_{2.5}$ exposure and cardiovascular hospitalization in more urban areas, though larger associations in less urban areas for respiratory hospital admissions. In the state of Georgia, Strickland et al. (2015) observed positive associations in less urban areas compared to null or negative associations in large metropolitan areas for less frequent respiratory hospital admissions, such as bronchitis, pneumonia, and sinusitis, though estimates were imprecise (i.e., wide 95% confidence intervals). Among more frequent respiratory outcomes, such as asthma, there was less evidence of effect modification. In contrast to other studies of respiratory outcomes, there was a trend of stronger associations for respiratory hospital admissions in urban areas in Southern California, compared to less urban counties in the Central Valley (Yap et al., 2013).

In studies of reproductive outcomes, Hu et al. (2015) observed an increased risk of gestational diabetes in Florida for mothers in rural areas. Meanwhile, in a nationwide study of infant births in Canada, Stieb et al. (2015) observed no substantial evidence of modification by maternal residential status. However, the authors observed small increases in rural births at risk for small for gestational age, as well as a decline in term birthweight.

12.5.5.2 Residential Proximity to Traffic

Traffic-related air pollution is a complex mixture typically consisting of both particulate and gaseous pollutants. Elevated near-road concentrations of UFP have been observed, although measured $PM_{2.5}$ concentrations are generally not elevated near the road (Karner et al., 2010), given that most $PM_{2.5}$ is produced via atmospheric chemistry. Both traffic-related air and noise pollution have been hypothesized to be associated with detrimental health effects; however, few studies have examined if

residential traffic proximity modifies existing associations between short- and long-term $PM_{2.5}$ exposure and health effects. No studies examined residential proximity to traffic in the 2009 PM ISA, though one study did suggest urban areas of low SES were disproportionately exposed to traffic-related pollutants (Yanosky et al., 2008).

Recent epidemiologic studies provide limited evidence that those living close to major roadways may be at greater risk for $PM_{2.5}$ associated cardiovascular or respiratory effects compared to those living farther from major roadways. In a study of short-term $PM_{2.5}$ exposure using data from the multicity MESA cohort, Auchincloss et al. (2008) observed stronger positive associations with pulse pressure and systolic blood pressure among those living within 300 meters of highways compared to those living further from highways, as well as positive associations for those in areas of higher road density. Smaller studies also observed stronger associations with $PM_{2.5}$ among residents living close to major roadways; however, the evaluated distance from roadways varied. In Atlanta, Georgia Sinclair et al. (2014) observed higher risk for $PM_{2.5}$-related asthma pediatric primary care visits among residents within 150 meters of major roadways, though not at 300 meters. Among stroke hospitalizations in southern Israel, Yitshak Sade et al. (2015) observed an increased risk of ischemic stroke for those living within 75 meters from main roads (OR: 1.42, 95% CI: 1.06, 1.87) compared to those further than 75 meters away (OR: 1.06, 95% CI: 0.89, 1.27).

A limited number of animal toxicology studies also support the importance of proximity to PM source. In Los Angeles, the enhancement of allergic responses was greater in allergic BALB/c mice exposed to $PM_{2.5}$ CAPs (multiday, 400 $\mu g/m^3$) 50 m from a busy roadway compared to those at a distance of 150 m (Kleinman et al., 2005). Additionally, a single acute exposure to aerosolized diesel exhaust particles resulted in increased BALF IL-4 levels in OVA-sensitized/challenged mice at exposures of 2000 $\mu g/m^3$, but not 870 $\mu g/m^3$ (Farraj et al., 2006a, b).

Summary

Overall, there is inadequate evidence to determine if residential location, either close proximity to a roadway or in a rural or urban area, increases risk for $PM_{2.5}$-related health effects. There is evidence that degree of urbanicity may modify the risk of $PM_{2.5}$-related health effects, particularly from large nationwide studies of mortality and long-term $PM_{2.5}$ exposure; however, in contrast to studies of mortality, several cardiovascular, respiratory, reproductive, and developmental studies observed limited evidence of increased risk in rural areas compared to urban areas. There may also be differences between metro areas of different sizes, though interpreting these trends is limited by the varying definition of urbanicity across studies. Furthermore, despite recent developments in methods to estimate ambient $PM_{2.5}$ concentrations, the limited availability of monitored data in smaller metropolitan and rural locations to validate modeled ambient $PM_{2.5}$ remains an important limitation (Sections 3.3.2, 3.3.3, 3.4.2.4). A limited number of epidemiologic studies also provide some evidence of stronger $PM_{2.5}$ related effects for those living closer to major roadways for asthma, stroke, and elevated

blood pressure compared to those living further from roadways. The available animal toxicology studies also suggest elevated immune responses among mice exposed to traffic-related exhaust. However, there is insufficient information available to determine how far these effects may extend from roadways, and if the relevant distances vary by health outcome, or other factors, such as levels of noise pollution.

12.6 Behavioral and Other Factors

12.6.1 Smoking

Overview

- It is unclear whether smoking exacerbates health effects associated with air pollutant exposures, including PM, and the potential for this was not evaluated in the 2009 PM ISA.
- Recent evidence does not indicate that smoking modifies the effect of long-term $PM_{2.5}$ exposures on cardiovascular disease or mortality; evidence evaluating differential effects by smoking status is limited for short-term $PM_{2.5}$ exposures.
- **Overall, the evidence is inadequate to determine whether individuals who smoke are at increased risk of $PM_{2.5}$-related health effects compared to those that do not smoke.**

Smoking is a common behavior as indicated by the 2016 National Health Interview Survey which estimated that within the U.S. adult population approximately 15.5% of individuals report being current smokers and 21.5% report being a former smoker (Blackwell and Villarroel, 2018). Smoking is a well-documented risk factor for many diseases, but it is unclear whether smoking exacerbates health effects associated with air pollutant exposures, including PM.

A number of studies have evaluated whether smoking status modifies the relationship between $PM_{2.5}$ exposure and health effects. The majority of these studies examined the relationship between long-term $PM_{2.5}$ exposure and mortality or cardiovascular morbidity. Generally, little difference is observed in the relationship between long-term exposure to $PM_{2.5}$ and mortality or cardiovascular morbidity when examined by smoking status. When differences in the relationship do occur, there is no consistent pattern or trend that support current, former, or ever smokers (i.e., both current and former smokers) being at increased or decreased risk than never smokers for these health outcomes. In a reanalysis of the ACS cohort, Turner et al. (2017) evaluated the interaction between $PM_{2.5}$ exposure and smoking, stratifying $PM_{2.5}$ exposure into low (<10.59 $\mu g/m^3$) and high (>14.44 $\mu g/m^3$) categories. These authors observed positive associations between higher $PM_{2.5}$ exposures and both total and CVD mortality; the interaction between current smoking and high $PM_{2.5}$ exposure increased the risk by 10%. In addition to the mortality and cardiovascular effects, several studies examined the ability of smoking status to modify the relationship between long-term $PM_{2.5}$ exposure and changes in blood pressure (Chan et al.,

2015; Mu et al., 2014; Fuks et al., 2011; Auchincloss et al., 2008) and indicators of atherosclerosis (Bauer et al., 2010; Lenters et al., 2010) and observed no consistent pattern among any smoking strata.

A smaller number of studies examined smoking status as a potential modifier of the effect of short-term $PM_{2.5}$ exposure on health outcomes (Supplemental Table S12-14) (U.S. EPA, 2018). A multicity analysis of mortality observed higher effects of $PM_{2.5}$ in counties where the prevalence of smoking was higher, but lacked individual-level smoking data (Dai et al., 2014). O'Donnell et al. (2011) examined whether the relationship between short-term $PM_{2.5}$ exposure and ischemic stroke differed by smoking status of participants and observed no evidence that smoking modified this relationship.

Overall, the inconsistent evidence is inadequate to determine whether individuals who smoke are at increased risk of $PM_{2.5}$-related health effects compared to those that do not smoke. A number of long-term exposure studies observed a mix of positive or nearly null associations for mortality and cardiovascular morbidity endpoints, but no clear or consistent trend is apparent among current, former, or ever smokers when compared to never smokers. Fewer studies evaluated smoking as an effect modifier of the relationship between short-term $PM_{2.5}$ exposure and health outcomes, and one study observed a stronger $PM_{2.5}$-mortality relationship in counties with a higher prevalence of smoking, but no individual-level data were available. Additionally, the varied metrics used to define smoking across studies (e.g., current, former, quantity) is a particular uncertainty in this evidence base.

12.6.2 Diet

Overview

- Dietary habits are well-established risk factors for metabolic/cardiovascular conditions that may be associated with $PM_{2.5}$ exposure; diet is an important source of anti-inflammatory and antioxidant compounds that may alter early biological responses to $PM_{2.5}$.
- Limited stratified epidemiologic analyses of alcohol or fruit and vegetable consumption do not indicate differences in mortality and $PM_{2.5}$ exposure.
- Limited evidence from controlled human exposure studies in the current and previous ISA demonstrates reduced cardiovascular and inflammatory responses among those taking B vitamin supplements
- **Overall, the evidence is inadequate to determine whether dietary patterns modify $PM_{2.5}$-related health effects.**

Dietary habits are well established risk factors for a variety of health outcomes, in particular, the development of metabolic-related conditions that may simultaneously be associated with PM exposure (Cardiovascular Effects, Section 6.2.1 and Section 6.3.1; Metabolic Effects, Section 7.2.1). It is possible that as dietary habits influence the development of chronic disease, there are increased risks of other $PM_{2.5}$-health effects for those with cardiovascular disease (Section 12.3.1), diabetes (Section 12.3.2), and

obesity (Section 12.3.3). Dietary tendencies also differ across the U.S. population, for example, low socioeconomic status (SES) individuals may have limited access to fresh foods (Larson et al., 2009). Limited access to fresh foods may lead to reduced intake of anti-inflammatory compounds and antioxidant polyunsaturated fatty acids and vitamins, which has been hypothesized to increase a population's risk of developing a PM-related health effect (Romieu et al., 2005).

The 2009 PM ISA concluded that nutritional status, among other surrogates of SES, may modify the association between PM and various health outcomes. Evidence for this conclusion was largely based a single study that examined $PM_{2.5}$ exposure and heart-rate variability (HRV) by nutritional status among those with genetic predisposition for cardiovascular disease (Baccarelli et al., 2008). The authors found that when individuals with genetic polymorphisms increased their consumption of B vitamins or methionine, they no longer observed an association between $PM_{2.5}$ and HRV. More recently, several studies have evaluated the ability of alcohol, fruit and vegetable consumption, and fatty acid supplementation to modify associations between $PM_{2.5}$ exposure and health outcomes in populations beyond those with specific genetic polymorphisms, primarily for long-term $PM_{2.5}$ exposure and mortality. While some studies observed differential effects, there is little consistency across studies, and effect estimates were often imprecise (i.e., wide 95% confidence intervals) (Supplemental Table S12-15) (U.S. EPA, 2018).

A limited number of epidemiologic studies evaluated effect measure modification by alcohol consumption. In a study of the Canadian Community Health Survey cohort, Pinault et al. (2016) examined associations between mortality and long-term $PM_{2.5}$ exposure and observed little evidence of differences based on regular drinking status for all-cause, cardiovascular, or respiratory mortality. In a study of long-term $PM_{2.5}$ exposure and systemic inflammation among mid-life women, Ostro et al. (2014) also observed little difference in C-reactive protein changes between abstainers and occasional consumers of alcohol. However, in a subanalysis examining the probability of a clinically relevant level of CRP (3 mg/l), the authors observed a positive association in older women who abstained from alcohol compared to a null association among older women who were occasional drinkers.

Several epidemiologic studies that examined the association between mortality and long-term $PM_{2.5}$ exposure evaluated potential modification by fruit and/or vegetable consumption patterns. Overall, few differences in mortality were observed when results were stratified by dietary patterns, and there is no consistent pattern to support greater fruit and vegetable consumptions leads to differential risk compared to lower fruit and vegetable consumption. U.S. based studies of cardiovascular mortality (Pope et al., 2014) and lung cancer mortality (Turner et al., 2011) did not observe a consistent pattern of differential risk by diet. Results stratified by quartile of fat consumption showed a similar pattern as when stratifying by fruit and vegetable consumption (Pope et al., 2014). Using data from the Canadian Community Health Survey cohort, Pinault et al. (2016) observed similar inconsistencies by mortality type, where the risk of $PM_{2.5}$ associated mortality slightly increased, or decreased depending on mortality categorization for the group consuming at least five or more servings of vegetables and fruit per day. Likewise, in a pooled

analysis of mortality and long-term $PM_{2.5}$ exposure across European cohorts (ESCAPE), no consistent pattern was observed between groups based on estimated grams of fruit consumed per day for all-cause, cardiovascular mortality, or respiratory mortality (Beelen et al., 2014a; Beelen et al., 2014b; Dimakopoulou et al., 2014).

The 2009 PM ISA examined a single study on nutritional status, which observed B vitamin supplementation attenuated the association between $PM_{2.5}$ and HRV among individuals with specific genetic polymorphisms that are associated with increased cardiovascular risk (Baccarelli et al., 2008). A recent pilot crossover study using 2 hour CAPS exposures examined B vitamin supplementation in a more general population and continues to provide limited evidence that B vitamins may protect against subclinical cardiovascular and inflammatory responses. Zhong et al. (2017a) observed attenuation in effects for measure of HRV and inflammatory blood markers, while using the same study population Zhong et al. (2017b) observed attenuated effects for DNA methylation and mitochondrial DNA content following vitamin B supplementation.

Controlled human exposure studies among the elderly have also examined the role of fish and olive oil supplementation and provide limited evidence that these oils may protect against certain subclinical responses to short-term $PM_{2.5}$ CAPs exposure. A series of CAPs studies in Chapel Hill, North Carolina provided olive oil (OO) or fish oil (FO) supplements to participants for four weeks, and then examined cardiovascular responses after two hours of CAPs exposure (Section 6.2.6, Table 6-12 and Section 6.2.4, Table 6-9). Tong et al. (2015) observed larger changes in endothelial function (i.e., decreased flow-mediated dilation) in the FO and nonsupplemented groups compared to the OO group, as well as increased vasoconstrictor concentrations (i.e., endothelin-1) for the nonsupplemented group. Results of fibrinolysis were less consistent, with increased tissue plasminogen activator, but decreases D-dimer levels after 20 hours in the OO group, but not FO or nonsupplemented group. In examining electrophysiological responses, Tong et al. (2012) did not include a nonoil supplement group, though the authors observed decreased responses to CAP exposure for heart rate variability, QT repolarization, and some blood lipids, such as VLDL and triglycerides, among those using FO compared to those using OO.

Overall, there is inadequate evidence to determine whether dietary patterns modify $PM_{2.5}$-associated health effects. Based on the limited number of epidemiologic studies, there is little evidence of differences in the relationship between mortality and $PM_{2.5}$ based on either alcohol or fruit and vegetable consumption. However, controlled human exposure studies of B vitamin, fish, and olive oil supplementation suggest potential protective effects against short-term exposure to concentrated ambient particles. Among epidemiologic studies, the reliance on long-term mortality studies is an important limitation, as self-reporting biases may still be problematic in accurate collection of dietary habits.

12.7 Conclusions

This chapter characterized the evidence for factors that may result in populations and lifestages being at increased risk for $PM_{2.5}$-related health effects (Table 12-3). The evaluation of each factor focused on the consistency, coherence, and biological plausibility of evidence integrated across a range of scientific disciplines informing whether a specific population or lifestage might be at increased risk of a PM-related health effect using the systematic framework detailed in Table 12-1. In the evaluation and characterization of the evidence consideration was given to exposure, dosimetry, biological plausibility, and/or the relationships of PM exposure with health effects as evaluated in Chapters 5-11 of this ISA. As noted in the introduction to this chapter, the 2009 PM ISA focused broadly on the extent to which evidence indicated that certain populations or lifestages were "susceptible" to PM-related health effects, but more recent ISAs have applied the systematic framework so the evaluation and conclusions in this ISA are more nuanced. Table 12-3 presents a summary of the conclusions and evidence evaluated and integrated in this chapter for each factor potentially resulting in an increase in risk for a $PM_{2.5}$-related health effect.

Of the factors considered, race and lifestage (children) were the only factors for which evidence was adequate to indicate an increase in risk for $PM_{2.5}$-related health effects (Section 12.5.4 and Section 12.5.1.1). In particular, evidence for both health effects, primarily mortality, and exposure demonstrate that nonwhite populations are at increased risk compared to whites. Several high-quality studies indicate that nonwhite populations across different geographical regions are exposed to higher concentrations of $PM_{2.5}$. In addition, a number of high-quality epidemiologic studies demonstrate stronger associations in nonwhite populations for $PM_{2.5}$-associated mortality. Increased risk for nonwhites compared to whites has also been demonstrated for other health outcomes including respiratory and cardiovascular effects and birth outcomes, but there is less confidence in the evidence for these outcomes.

There is strong evidence from studies examining health endpoints specific to children indicating that children are at increased risk to the effects of $PM_{2.5}$ exposure. Specifically, epidemiologic studies of long-term $PM_{2.5}$ exposure demonstrate associations with impaired lung function growth (Section 5.2.2.1.1), decrements in lung function (Section 5.2.2.2.1), and increased incidence of asthma development in children (Section 5.2.3.1). The evidence from stratified analyses provides limited direct evidence that children are at increased risk of $PM_{2.5}$-related health effects compared to adults. In addition, there is some evidence indicating that children can have higher $PM_{2.5}$ exposures than adults and that there are dosimetric differences in children compared to adults that can contribute to higher doses.

There is suggestive evidence that populations with pre-existing cardiovascular or respiratory disease (Section 12.3.1 and Section 12.3.5), populations that are overweight or obese (Section 12.3.3), populations that have particular genetic variants (Section 12.4), or populations that are of low SES (Section 12.5.3) are at increased-risk for $PM_{2.5}$-related health effects compared to respective reference populations. While stratified analyses for pre-existing cardiovascular disease do not consistently indicate

differential risk, there is some evidence that those with hypertension may be at increased risk compared to those without hypertension. In addition, there is strong evidence supporting a causal relationship between exposure to $PM_{2.5}$ and cardiovascular health effects, particularly mortality and ischemic heart disease (Chapter 6), and those with underlying cardiovascular conditions related to these serious outcomes may be at increased risk based on pathophysiological considerations compared to those without these conditions. Similarly, for pre-existing respiratory disease, evidence is limited that directly informs differential risk between those with and without pre-existing respiratory disease. However, Chapter 5 concluded that there is likely to be a causal relationship between short-term $PM_{2.5}$ exposure and respiratory effects, based primarily on evidence for exacerbation of asthma and COPD. Those with pre-existing obesity may also be at increased risk compared to those of healthy weight, based on evidence indicating greater risk for mortality associated with long-term exposures to $PM_{2.5}$ in individuals who are obese or overweight compared to those who are normal weight. In considering the evidence for genetic background, a variety of gene variants have been studied. There is a consistent trend for increased risk for respiratory and cardiovascular effects associated with $PM_{2.5}$ across gene variants involved in the glutathione pathway and oxidant metabolism, which is consistent with biological plausibility indicating that oxidative stress is an early biological response to $PM_{2.5}$ exposure. Evidence for other genetic variants is very limited. Finally, evidence indicates that those that are of low SES are more likely to have higher $PM_{2.5}$ exposures and that low SES, as measured by metrics for income, may increase risk for $PM_{2.5}$-associated mortality compared to higher SES categories, though there is some inconsistency in the evidence and heterogeneity in the metrics used.

There is inadequate evidence to determine whether pre-existing diabetes (Section 12.3.2), elevated cholesterol (Section 12.3.4), lifestage: older adults (Section 12.5.1.2), residential location (including proximity to source and urban residence [Section 12.5.5], sex [Section 12.5.2], or diet [Section 12.6.2]) modify risk for $PM_{2.5}$-associated health effects. For lifestage related to older adults (Section 12.5.1.2) there is limited evidence indicating that older adults are at increased risk for $PM_{2.5}$-related health effects; however, epidemiologic panel studies and controlled human exposure studies of older adults provide some evidence that subclinical cardiovascular outcomes are associated with short-term exposure to $PM_{2.5}$ for this lifestage. Evidence for other factors is inadequate due to limited evidence (residential patterns, diet) or inconsistency across the available evidence (diabetes and sex).

Table 12-3 Summary of evidence for populations potentially at increased risk of $PM_{2.5}$-related health effects.

Evidence Classification	Factor Evaluated	Population/Lifestage Potentially at Increased Risk	Factor-specific Evidence	Evidence Informing an Increase in Risk
Adequate Evidence	Race (Section 12.5.4)	Nonwhite populations		Evidence from multiple high-quality studies demonstrating higher $PM_{2.5}$ exposure in nonwhite populations. Consistent evidence from high quality studies demonstrating increased risk for mortality and cardiovascular/respiratory morbidity.
	Lifestage	Children (Section 12.5.1.1)	Strong evidence demonstrating health effects in children, particularly from epidemiologic studies of long-term $PM_{2.5}$ exposure and impaired lung function growth, decrements in lung function, and asthma development.	Limited evidence from stratified analyses to inform increased risk in children compared to adults. However, evidence from studies of pediatric asthma and impaired lung development provide strong and consistent evidence that effects are observed in children.
Suggestive Evidence	Pre-existing Disease	Pre-existing Cardiovascular Disease (Section 12.3.1)	Causal relationship for $PM_{2.5}$ exposure and cardiovascular effects based on CV mortality and morbidities that are plausibly more prevalent in those with pre-existing CV disease/conditions.	Generally supportive evidence from epidemiologic studies demonstrating differential effects for those with hypertension. Limited and inconsistent evidence for other pre-existing cardiovascular diseases.
		Pre-existing Respiratory Disease (Section 12.3.5)	Likely to be causal relationship for short-term $PM_{2.5}$ exposure and respiratory effects based primarily on evidence for asthma and COPD exacerbation. Evaluated outcomes are often specific to those with asthma or COPD and those without asthma or COPD are not included for comparison.	Limited evidence. Primarily cardiovascular outcomes in epidemiologic studies. Although asthma exacerbation is a key outcome for conclusions on respiratory effects, no informs an increase in risk for those with asthma compared to those without. There is very limited evidence for COPD.

Table 12-3 (Continued): Summary of evidence for potential increased risk of $PM_{2.5}$-related health effects

Evidence Classification	Factor Evaluated	Population/Lifestage Potentially at Increased Risk	Factor-specific Evidence	Evidence Informing an Increase in Risk
Suggestive Evidence (continued)	Pre-existing Disease (continued)	Obesity (Section 12.3.3)		Based primarily on evidence for increased risk for mortality with supporting evidence from studies of subclinical cardiovascular outcomes.
	Genetic background (Section 12.4)	Individuals with variant genotypes	Biological plausibility for $PM_{2.5}$-associated health effects is based on biological pathways including oxidative stress as early biological responses upon exposure to $PM_{2.5}$.	Generally consistent evidence for increased risk for respiratory and cardiovascular outcomes for genetic variants in the glutathione pathway, which has an important role in oxidative stress. Limited evidence for other genetic variants.
Suggestive Evidence (Continued)	Socioeconomic Status (Section 12.5.3)	Low socioeconomic status		Evidence demonstrates increased exposure and some evidence for stronger associations for mortality with low SES. Comparison across SES metrics are a limitation.
Inadequate Evidence	Pre-existing disease	Pre-existing diabetes		Inconsistent evidence across studies of mortality, cardiovascular morbidity, and inflammation.
	Lifestage	Older adults (Section 12.5.1.2)	Evidence demonstrating health effects in older adults, particularly from short- and long-term $PM_{2.5}$ exposure and cardiovascular or respiratory hospital admission, emergency department visits, or mortality.	Inconsistent evidence across a large body of studies with stratified analyses.
	Residential location (Section 12.5.5)	Near-road or urban residence		Some evidence demonstrates potential for urbanicity to modify $PM_{2.5}$-related health effects, but results are inconsistent across the broad range of metrics used.
	Sex (Section 12.5.2)	Males[a]	Males: Reproductive factors e.g., sperm motility. Females: Gestation and birth outcomes.	Inconsistent evidence across studies for mortality and cardiovascular and respiratory effects.

Table 12-3 (Continued): Summary of evidence for potential increased risk of $PM_{2.5}$-related health effects

Evidence Classification	Factor Evaluated	Population/Lifestage Potentially at Increased Risk	Factor-specific Evidence	Evidence Informing an Increase in Risk
Inadequate Evidence (continued)	Smoking (Section 12.6.1)	Current smoking		Inconsistent evidence for modification of associations between $PM_{2.5}$ and mortality, cardiovascular, reproductive, metabolic, and reproductive outcomes.
Inadequate Evidence (Continued)	Diet (Section 12.6.2)	Individuals with reduced fruit/vegetable intake, alcohol consumption, or elevated cholesterol		Inconsistent evidence across a limited evidence base.
Evidence of no effect	None			

ISA = Integrated Science Assessment.

Males selected as potential at-risk group due to shorter life-span. The use of males or females as the reference/comparison group does not change the evaluation of evidence in determining differential risk.

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CHAPTER 13 WELFARE EFFECTS

Summary of Causality Determinations for Particulate Matter (PM) and Welfare Effects

This chapter characterizes the scientific evidence that supports causality determinations for PM exposure and nonecological welfare effects. The types of studies evaluated within this chapter are consistent with the overall scope of the ISA as detailed in the Preface (see Section P 3.1). More details on the causal framework used to reach these conclusions are included in the Preamble to the ISA (U.S. EPA, 2015).

Effect	Causality Determination
Visibility	Causal
Climate	Causal
Materials	Causal

13.1 Introduction

This chapter serves as the scientific foundation for the review of the secondary (welfare-based) National Ambient Air Quality Standards (NAAQS) for PM. The Clean Air Act definition of welfare effects includes, but is not limited to, effects on soils, water, wildlife, vegetation, visibility, weather, and climate, as well as effects on man-made materials, economic values, and personal comfort and well-being (CAA, 2005). In this review of the PM secondary NAAQS, welfare effects to be considered include PM-related visibility (Section 13.2), climate effects (Section 13.3) and materials damage and soiling (Section 13.4). As noted in the Preface , in the case of materials effects, the impacts of gaseous and particulate N and S wet deposition cannot be clearly distinguished, so both are considered in this review. The ecological effects associated with the deposition of oxides of nitrogen, oxides of sulfur and PM are being addressed in a separate review [i.e., the Integrated Science Assessment (ISA) for Oxides of Nitrogen, Oxides of Sulfur, and Particulate Matter-Ecological Criteria-(U.S. EPA, 2018)]. These PM-related ecological effects include nutrient enrichment, acidification, and sulfur enrichment associated with particle deposition, and the direct and indirect effects of PM on vegetation, soils, and biota.

The 2009 Integrated Science Assessment for Particulate Matter (2009 PM ISA) concluded that a causal relationship exists between PM and visibility impairment. Recent research provides additional evidence evaluated in the 2009 PM ISA, and confirms that a causal relationship exists between PM and visibility impairment. New research provides a better understanding of the relationship between PM composition and atmospheric visibility during a period of changing PM composition due to reduced

emissions of PM precursors. New research also indicates long-term visibility improvements throughout the U.S. There continues to be considerable uncertainty around quantifying acceptable visibility. Overall, the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment.

The 2009 ISA concluded that a causal relationship exists between PM and climate effects—specifically on the radiative forcing of the climate system, including both direct effects of PM on radiative forcing and indirect effects involving cloud processes. Recent research reinforces and strengthens the evidence evaluated in the 2009 PM ISA, and reaffirms that a causal relationship exists between PM and climate effects. This causality determination provides greater specificity about the details of these radiative forcing effects and increased understanding of additional climate impacts driven by PM radiative effects. The IPCC AR states that "Climate-relevant aerosol processes are better understood, and climate-relevant aerosol properties better observed, than at the time of AR4 [released in 2007]" (Boucher, 2013). Research since the 2009 PM ISA has also improved characterization of the key sources of uncertainty in estimating PM climate effects, particularly with respect to PM-cloud interactions. Substantial uncertainties, however, still remain with respect to key processes linking PM and climate, both because of the small scale of PM-relevant cloud microphysical processes compared to the resolution of state-of-the-art models, and because of the complex cascade of indirect impacts and feedbacks in the climate system that result from a given initial radiative perturbation caused by PM. These uncertainties continue to limit the precision with which these effects can be quantified. Despite these remaining uncertainties, though, overall the evidence is sufficient to conclude that a causal relationship exists between PM and climate effects.

The 2009 PM ISA (U.S. EPA, 2009) concluded a causal relationship between PM and effects on materials. For most topics related to materials damage, the fundamental understanding of mechanisms of soiling and corrosion has not changed; rather, additional studies lend further support to the findings from the previous ISA and effects on some materials have been further characterized. There is new information for glass and metals including modeling of glass soiling and identifying which pollutants are most influential in metal corrosion in a multipollutant environment, and how that varies between metals. Development of quantitative dose-response relationships and damage functions for materials besides stone has also progressed, with new dose-response curves published for glass, and a new summary of available materials damage functions. Since the 2009 ISA there is a growing body of research, including quantitative assessment, of PM impacts on the energy yield from photovoltaic systems.

13.2 Effects on Visibility

13.2.1 Introduction

The 2009 PM ISA concluded that "a causal relationship exists between PM and visibility impairment" based on strong and consistent evidence that PM is the overwhelming source of visibility impairment in both urban and remote areas (U.S. EPA, 2009). Visibility refers to the visual quality of the view, or scene, with respect to color rendition and contrast definition. It is the ability to perceive landscape form, colors, and textures. Visibility involves optical and physical processes of light interacting with scenic elements and the atmosphere, as well as psychophysical processes involving human perception, judgment, and interpretation. On very clear days, near objects have bright, crisp colors and textures while objects over 200 km away may still be visible. Even when there are no distant objects, a clear day produces vibrant blue skies and bright white clouds with sharp edges. Removal and addition of visible light to an observer's sight path reduces both the contrast of near objects and the ability to see distant objects. Light between the observer and the object can be scattered into or out of the sight path and absorbed by PM or gases in the sight path. The sum of scattering and absorption of visible light due to PM and gases is referred to as light extinction, b_{ext}.

In polluted environments, light extinction by gases is usually small compared to PM (Malm, 2016; U.S. EPA, 2009). Light absorbing carbon (e.g., soot and smoke), incorporating and often referred to as elemental, black, and brown carbon (Andreae and Gelencsér, 2006), and some crustal minerals (Moosmueller et al., 2012) are the only commonly occurring PM components that absorb light. However, all particles scatter light, and scattering by particles is usually greater than absorption by particles or than scattering or absorption by gases (Hand et al., 2011). Particulate scattering is dependent on particle shape, refractive index, and size. Provided these properties are known, light scattering can be accurately calculated for a distribution of particles.

The linkage between PM and human perception of haze[84] involves a number of physical/chemical/optical and psychophysical processes. These processes can be divided into three broad categories, around which the discussion of the evidence is largely organized: 1) the impairment of visibility by haze; 2) the spatial, temporal, and compositional distributions of PM and their optical properties causing the haze; and 3) human perception of and response to the haze.

Evidence in the 2009 PM ISA (U.S. EPA, 2009) supported that PM was the overwhelming source of visibility impairment in both urban and remote areas, and light scattering by gases contributed

[84]In Sections 13.2 and 13.3, the term haze is used as a qualitative description of the blockage of sunlight by dust, smoke, and pollution. This usage is widespread in the scientific literature on visibility and in discussion of the Regional Haze Rule (U.S. EPA, 2003). This contrasts with the use of the term haze in Section 13.4, where it is used as defined in the scientific literature on soiling of glass, i.e., the ratio of diffuse transmitted light to direct transmitted light (Lombardo et al., 2010).

substantially only under pristine conditions. Elemental carbon (EC) and some crustal minerals are the only common PM components that absorb light, and that light scattering is greatest for particles in the size range from 0.3 to 1.0 μm (U.S. EPA, 2009). The 2009 PM ISA (U.S. EPA, 2009) also described methods for estimating contributions of PM components to light extinction as well as direct optical measurements for light scattering, absorption, and total extinction (U.S. EPA, 2009). Particulate sulfate was found to be the dominant source (>40% of PM light extinction) of regional haze in the Eastern U.S. and an important contributor (>20% of PM extinction) elsewhere in the U.S. EC and organic carbon (OC) were found to be responsible for 10−40% the haze in the U.S., with the greatest contribution in the Northwest, although per unit mass sulfate had a greater impact on visibility because of its hygroscopicity. Particulate nitrate was found to be a substantial contributor in the Midwest and California and crustal material was an important contributor in the Southwest (U.S. EPA, 2009). Human perception of visibility impairment was also reviewed in the 2009 PM ISA (U.S. EPA, 2009) based on estimates of median acceptable values from existing visibility preference studies.

The discussion of PM visibility impairment opens with reviews of metrics and monitoring methods and approaches used for evaluating visual air quality and advances in their development (Section 13.2.2). The relationship between PM and visibility impairment, including the central role of mass scattering efficiencies and advances in their use to estimate atmospheric light extinction from network PM data are then described (Section 13.2.3). Next, recent PM network data are examined to provide an up to date summary of spatial and temporal visibility patterns (Section 13.2.4). Finally, reviews of new approaches to evaluating human perception and preferences concerning atmospheric visibility and its value are provided (Section 13.2.5).

13.2.2 Visibility Impairment

13.2.2.1 Visibility Metrics

Two fundamental characteristics of atmospheric visibility impairment are 1) a reduction in *visual range*, the greatest distance through the atmosphere at which a prominent object can be identified, and 2) a reduction in *contrast*, the sharpness with which an object can be distinguished from another object or background (Malm, 2016). Both of these concepts can be understood in terms of an *atmospheric extinction coefficient* that relates the distance of an observed object to atmospheric light extinction following the Beer-Lambert Law (Finlayson-Pitts and Pitts, 2000).

The atmospheric extinction coefficient (b_{ext}) is a measure of the alteration of radiant energy as it passes through the atmosphere. b_{ext} can be expressed as the sum of light scattering by particles (b_{sp}), scattering by gases, known as Rayleigh scattering (b_{sg}), absorption by particles (b_{ap}), and absorption by gases (b_{ag}):

$$b_{ext} = b_{sp} + b_{sg} + b_{ap} + b_{ag}$$

Equation 13-1

b_{ext} varies with concentration and composition of scattering and absorbing substances in the atmosphere, and is especially useful for relating visual properties of distant objects theoretically to known concentrations and characteristics of atmospheric species (Malm, 2016). According to Malm (2016). Consequently, as described in Section 13.2.1, light extinction by gases is usually small compared to PM, and b_{sp} and b_{ap} are the main contributors to b_{ext}.

Contrast and visual range can both be conceptualized in terms of b_{ext}. The contrast can be between a haze layer and its background or between two different elements within a landscape feature, referred to as contiguous contrast. Contrast can be expressed in terms of a single color or as a color contrast. *Threshold contrast* is the reduction of contrast between two features to a point where it can just be seen (Malm, 2016). A *suprathreshold* value is a contrast change that is just noticeable when a landscape feature is clearly visible (Malm, 2016). If the background is the sky and light is uniform then contrast follows the Koschmieder relationship $C_r = C_o T_r$, (Middleton, 1968; Koschmieder, 1924), where T_r is the transmittance over path length r (Malm, 2016). The uniform sky light conditions necessary for the Koschmieder relationship to be valid do not always hold, but are most likely to be met under hazy conditions (Malm, 2016). If uniform light conditions are met, the Koschmieder relationship works well for perceptibility of isolated scenic elements, but uncertainty increases as light conditions become less uniform. Also, contrast is a scene-dependent metric based on the perception of a single object, and may not be representative of responses to visual characteristics of the scenic view as a whole (Malm, 2016). This limits its use for comparing visual impairment between different scenes or locations. Still, the Koschmieder relationship is widely used for assessing atmospheric visibility impairment, including the explanation of visual range.

If a just visible black object is viewed against the sky and the sky radiances at the observer and landscape feature are equal, then the Koschmeider relationship can be used to define the visual range as

$$V_r = \frac{-ln(\varepsilon)}{\bar{b}_{ext}}$$

Equation 13-2

where ε is the threshold contrast (a contrast level that can just be detected). If $\bar{b}_{ext} = b_{ext}$ and the threshold contrast ε is taken to be 0.02 based on historical observations (Malm, 2016), visual range can be calculated from b_{ext}:

$$V_r = \frac{3.912}{b_{ext}}$$

Equation 13-3

If b_{ext} is constructed such that Rayleigh scattering, i.e., b_{sg}, is set equal to 10 Mm^{-1}, then V_r is known as the standard visual range (SVR), which by Equation 13-3 is 391 km.

Visual range and extinction coefficient are metrics that can be consistently measured and used to assess visual air quality and track its changes and responses to emissions and PM. A third widely used metric the deciview haze index is a log transformation of light extinction (Pitchford and Malm, 1994):

$$dv = 10\left(ln\frac{b_{ext}}{0.01\ km^{-1}}\right)$$

Equation 13-4

The deciview is similar to the decibel for acoustic measurements. A one deciview (dv) change is about a 10% change in light extinction, which is a small change that is detectable for sensitive viewing situations. The haze index in deciview units is an appropriate metric for expressing the extent of haze changes where the perceptibility of the change is an issue. The Regional Haze Rule has adopted the deciview haze index as the metric for tracking long-term haze trends of visibility-protected federal lands (U.S. EPA, 2001).

Due to the dependence of the perception of haze by the human observer, scenic elements, and atmospheric optics, a number of different visibility metrics have been proposed over the years. They tend to fall into two broad categories: those metrics that are scene dependent, incorporating landscape characteristics and possibly human responses to the changes and those metrics that are independent of the scene but depend only on optical characteristics of the atmosphere, also called universal metrics.

Atmospheric extinction coefficient, visual range, and deciview are all universal, or scene independent metrics. There are also scene-dependent metrics, which incorporate changes in the radiance from landscape features and possibly human responses due to haze and depend on the landscape features, haze, illumination, and possibly the observer. Although these metrics are dependent on multiple scene features, it is also useful to have metrics that can directly relate human judgments of the visual air quality of a scene under varying haze conditions to a basic atmospheric variable such as light extinction. Contrast is a scene dependent metric. Numerous other universal and scene dependent metrics have been developed, but are not included in this assessment because they have not been used in studies reviewed here and were thoroughly reviewed recently (Malm, 2016).

13.2.2.2 Monitoring of Visibility Impairment

Direct PM light extinction, scattering, and absorption measurements are considered more accurate estimates derived from PM mass measurements because they do not depend on assumptions about particle characteristics (e.g., size, shape, density, component mixture, etc.). They can also be made with high time resolution, allowing characterization of subdaily temporal patterns of visibility impairment. Methods for measurement of light extinction, scattering, and absorption were reviewed in the 2009 PM ISA, which

included discussion of transmissometers for measurement of path-averaged light extinction and integrating nephelometers for measurement of light scattering. The use of integrating nephelometers for investigating effects of ambient PM size and water growth characteristics on light scattering was also described. The discussion also included measurement of PM light absorption by transmittance through filters on which PM has been collected as well as with aethelometers and photoacoustic instruments (U.S. EPA, 2009). Not reviewed in the 2009 PM ISA were methods for measuring scene-dependent visibility metrics that quantify the appearance of the view, accounting for the effects of particle and lighting conditions on the appearance of the scene. These include teleradiometers and telephotometers as well as photography and photographic modeling, which were described in the 2004 PM AQCD (U.S. EPA, 2004) and recently updated by Malm (2016). The discussion here is focused on strengths, limitations, and new developments of methods that were also discussed in the 2009 PM ISA (U.S. EPA, 2009), but includes recent research results that confirm or add to this body of knowledge. The convention for visibility monitoring is to make measurements at or near 550 nm, which is the wavelength of maximum eye response.

The integrating nephelometer was described in the 2009 PM ISA (U.S. EPA, 2009). It is characterized by high sensitivity and good sample control options and has been a widely used scattering instrument for air-quality-related visibility and PM monitoring purposes (Charlson et al., 1974). Integrating nephelometers significantly underestimate large particle scattering (Mueller et al., 2011b; Massoli et al., 2009; Mueller et al., 2009; Quirantes et al., 2008; Anderson and Ogren, 1998). Thus, they are better suited to measure scattering from fine PM than total or coarse PM. Historically, nephelometer chambers have been heated by radiation from their lamps and nearby electronics, drying out hygroscopic particles such as sulfates and nitrates underestimating ambient scattering. Current nephelometers generally use LED light sources, substantially reducing heating and its effects (Mueller et al., 2011b). Polar nephelometers measure the scattering as a function of scattering angle and thus can define the scattering phase function for a given aerosol (Dolgos and Martins, 2014; McCrowey et al., 2013). This can be important for visibility impairment assessments, since the path function will vary as a function of sun, landscape features, and observer geometry.

Forward and backscatter monitors measure light scattering in a prespecified solid angle (Heintzenberg, 1978). Open-air, forward scattering instruments are robust instruments and are extensively used by the National Weather Service (NWS) Automated Surface Observing System (ASOS) for characterizing visibility, principally for transportation safety purposes (NOAA, 1998; Richards et al., 1996). These instruments are also increasingly being used in Asian air quality and visibility studies, e.g., Shahzad et al. (2013) and Wang et al. (2014b).

Light absorption by PM is typically due mostly to black carbon (BC), with some contribution from organic matter also possible (Petzold et al., 2013). Soil or dust particles in the atmosphere also contribute to potentially significant amounts of atmospheric absorption (Fialho et al., 2014). Aerosol absorption measurements are made from a loaded filter based on the reflectance and transmittance of light

through the filter (Moosmüller et al., 2009; Bond et al., 1999) or in situ using a variety of methods including photoacoustic absorption spectrometry (Moosmüller et al., 2009).

All filter-based measurements require adjustments to the optical measurements to account for filter and sampled particle light-scattering effects associated with particles concentrated on and within the matrix of the filters (U.S. EPA, 2009; Bond et al., 1999). In a recent intercomparison of filter based absorption measurements, Mueller et al. (2011a) found a large variation in response from the different instruments and concluded that current correction functions for these measurements are not adequate. Quartz or glass fiber filters are the most widely used substrates in filter based absorption measurements. Organic gases are known to adsorb onto these filter media biasing organic carbon measurements, and these can be pyrolyzed to form artifact BC during the analysis, producing substantial biases in filter-based absorption measurement (Vecchi et al., 2014).

In situ measured absorption was also described in the 2009 PM ISA (U.S. EPA, 2009), and does not suffer from filter-based artifacts. Two first principle methods are absorption measured by extinction-minus-scattering and photoacoustic absorption spectrometry. The extinction-minus-scattering method suffers from potentially large subtraction errors for aerosols with high single scattering albedo and systematic errors such as the truncation errors in nephelometer scattering measurement for large particles (Singh et al., 2014; Moosmüller et al., 2009). Photoacoustic spectrometry operates by measuring the changes in pressure waves resulting from the heating and cooling of absorbing aerosols from a pulsed source of electromagnetic energy, typically a laser (Arnott et al., 2005; Arnott et al., 1999). These methods have been found to have low errors (Moosmüller et al., 2009). New developments include the combination of photoacoustic absorption measurements with integrating nephelometer in the same instrument package. For example, Sharma et al. (2013) developed a new multiwavelength, photoacoustic nephelometer spectrometer that measures scattering and absorption at wavelengths of 417, 475, 542, 607, and 675 nm.

Transmissometers measure the change in light intensity over a known distance from which b_{ext} can be derived. Long-path transmissometers were with path lengths up to 10 km were described in the 2009 PM ISA, and were concluded to suffer from a number of interferences that can cause large errors and difficulty in data interpretation (U.S. EPA, 2009; Debell et al., 2006). Cavity ring-down transmissometers do not suffer from these interferences. In this configuration, a beam of light, typically with wavelengths between 500 and 600 nm, is reflected back and forth between mirrors through an air sample, and the decay in the beam intensity over time is measured (Singh et al., 2014; Fiddler et al., 2009; Moosmuller et al., 2005; Wheeler et al., 1998). A disadvantage of the cavity ring-down configuration is that it is a point measurement and does not account for changes in b_{ext} over a sight path.

For scene-dependent visibility metrics, digital cameras have become used in much the same way as teleradiometers, recording signals proportional to radiance from all landscape features in the view. Digital or photographic cameras can be used to collect two-dimensional arrays, referred to as pixels, of film densities or digitized voltages in three color channels that are proportional to the image radiance

field, and if calibrated properly, provide quantitative radiance levels over the scene (Malm, 2016; Du et al., 2013). Advances have also been made in the application of photography in a less polluted environment. Most studies of visibility impairment have been carried out under fairly hazy conditions in urban environments, where fairly uniform lighting conditions correspond closely to conditions of the Koschmieder relationship. Furthermore, urban scenes tend to be gray, devoid of color associated with vegetation or brightly colored cliffs or terrain faces, such as those viewed in many of our national parks and wilderness areas. Bright edges of cloud formations are typically far enough from the observer to be obscured by heavy haze levels. Malm et al. (2015) investigated the ability to extract useful visibility metrics from routine webcams located in low-haze environments, specifically at the Grand Canyon National Park, Arizona, and Great Smoky Mountains National Park, Tennessee. This task is made more challenging by the effects of greater changes in lighting conditions that occur in low-haze conditions. Nonetheless, it was shown that meaningful relationships between metrics derived from the webcam images and atmospheric optical variables could be obtained as long as the indices were averaged over sufficient time to average out the effects of changing lighting.

13.2.3 Relationship between Particulate Matter and Visibility Impairment

Our understanding of the relationship between light extinction and PM mass has changed little since the 2009 PM ISA (U.S. EPA, 2009). Briefly, the impact of PM on light scattering depends on particle size and composition as well as relative humidity. All particles scatter light as described by Mie theory, which relates light scattering to particle size, shape, and index of refraction (Van de Hulst, 1981; Mie, 1908). Hygroscopic particles like ammonium sulfate, ammonium nitrate, and sea salt exhibit substantial growth as relative humidity increases, leading to increased light scattering (U.S. EPA, 2009). For externally mixed particles, a linear relationship between the b_{ext} is the sum of the mass concentration of each PM species multiplied by its specific mass extinction efficiency can be derived from Mie theory (Ouimette and Flagan, 1982):

$$b_{ext} = \sum_j a_j \, f_j(RH) M_j$$

Equation 13-5

where the species (j) mass concentration is given by M_j ($\mu g/m^3$); its extinction efficiency is given by α_j ($m^2 g^{-1}$); and its hygroscopic scattering growth factor given by $f_j(RH)$. The particle species j can be for a single compound or class of compound, such as particulate organic matter or even $PM_{2.5}$.

Equation 13-5 not only describes the theoretical relationship between light extinction and PM characteristics, but also provides the basis for practical use of mass scattering efficiencies in combination with ambient PM concentration data to estimate light extinction. This approach was previously described in the 2009 PM ISA (U.S. EPA, 2009), but is included here because it was used to estimate the extinction data used to examine seasonal and spatial patterns of visibility impairment in Section 13.2.4. Equation

13-5 strictly applies to external mixtures of PM, i.e., PM is composed of a mixture of species, but each single particle is composed of only one of species. Although ambient PM is usually a complex and unknown combination of both internal and external mixtures of PM components, differences in calculated light extinction using various external and internal mixture assumptions were generally less than about 10%. As a result, the form of Equation 13-5 has been accepted as a reasonable approach to apportioning light extinction to PM components (U.S. EPA, 2009).

Applying Equation 13-5 to major PM species generates Equation 13-6, which was developed specifically for use with PM monitoring data (Section 13.2.4) (U.S. EPA, 2009; Malm et al., 1994):

$$b_{ext} \cong 3f(RH)([AS] + [AN]) + 4[OM] + 10[EC] + 1[FS] + 0.6[CM] + 10$$

Equation 13-6

Light extinction (b_{ext}) is in units of Mm^{-1}; [AS], [AN], [OM], [EC], [FS], [CM] are the concentrations in $\mu g/m^3$ of ammonium sulfate, ammonium nitrate, organic matter, elemental carbon, fine soil, and coarse mass, respectively; f(RH) is the relative-humidity-dependent water growth function, and the various coefficients are empirically derived mass scattering and absorption coefficients originally proposed by (Malm et al., 1994). Particulate organic matter concentration [OM] is derived from measured organic carbon concentration [OC] by multiplying by a factor of 1.4, [OM] = 1.4 [OC]. Equation 13-6 is widely referred to as the *original IMPROVE algorithm* to distinguish it from subsequent variations developed later. Although considerable research has focused on evaluating mass extinction coefficients, assessing the linearity of the relationship, and investigating the need for additional terms, a modification of Equation 13-6 (Hand et al., 2011) remains widely used for relating light extinction to PM components, including this document. Three major modifications were made to the Equation 13-6 for use in the most recent IMPROVE network report (Hand et al., 2011):

- A sea salt term was added.
- The factor used to compute particulate organic matter concentration from organic carbon concentration was increased from [OM] = 1.4[OC] to [OM] = 1.8[OC].
- A site-specific term based on elevation and mean temperature was used for Rayleigh scattering (gas scattering) instead of the constant value of 10 Mm^{-1} used in the original equation for all sites.

The resulting equation has been referred to as the *modified original IMPROVE algorithm* to distinguish it other, more extensive revisions:

$$b_{ext} \cong 3f(RH)([AS] + [AN]) + 4[OM] + 10[EC] + 1[FS] + 1.7f(RH)[SS]$$

Equation 13-7

where [SS] is sea salt concentration. All estimates of light extinction from $PM_{2.5}$ species in this document were made with Equation 13-7.

13.2.3.1 Estimated Mass Extinction

Mass scattering efficiencies, α_{sp}, can be calculated for single particle components or composites of different particle types, e.g., $PM_{2.5}$. The three main methods for calculating mass scattering efficiencies, α_{sp}, are 1) as a simple ratio of measured mass concentrations to measured light scattering coefficients, 2) by multilinear regression with b_{ext} as the independent variable and the measured PM mass concentrations for each species as the dependent variables, and 3) from Mie theory (see Section 13.2.3.1) if PM distribution, chemical composition, and optical properties are known (Malm, 2016; U.S. EPA, 2009; Hand and Malm, 2007). Average dry mass scattering efficiencies estimated by various methods from ground-based measurements in a survey of 60 studies since 1990 by Hand and Malm (2007). Results were briefly discussed in the 2009 PM ISA (U.S. EPA, 2009) and are more fully presented in Table 13-1. The results for individual species were considered generally consistent with the coefficients of Equation 13-6 or Equation 13-7 (U.S. EPA, 2009).

Table 13-1 Mass scattering efficiencies for urban, remote, and ocean regions.

Species/Mode[a]	Urban (m^2/g)	Remote/Rural Continental (m^2/g)	Ocean/Marine (m^2/g)	All Methods (m^2/g)
Fine mixed	3.2 ± 1.3 (32)	3.1 ± 1.4 (24)	4.1 ± 0.8 (42)	3.6 ± 1.2 (98)
Coarse mixed	0.6 ± 0.3 (6)	0.7 ± 0.4 (24)	1.6 ± 1.0 (21)	1.0 ± 0.9 (51)
Total mixed	1.7 ± 1.0 (14)		2.5 ± 1.0 (6)	1.9 ± 1.1 (20)
Fine sulfate	2.6 ± 0.7 (9)	2.7 ± 0.5 (56)	2.0 ± 0.7 (28)	2.5 ± 0.6 (93)
Fine nitrate	2.2 ± 0.5 (6)	2.8 ± 0.5 (42)		2.7 ± 0.5 (48)
Fine POM	2.5 (1)	3.1 ± 0.8 (38)	5.6 ± 1.5 (19)	3.9 ± 1.5 (58)
Coarse POM			2.6 ± 1.1 (19)	2.6 ± 1.1 (19)
Total POM			3.5 ± 0.9 (8)	3.5 ± 1.0 (8)
Fine dust		2.6 ± 0.4 (4)	3.4 ± 0.5 (19)	3.3 ± 0.6 (23)
Coarse dust		0.5 ± 0.2 (3)	0.7 ± 0.2 (19)	0.7 ± 0.2 (22)
Total dust		0.71 (1)	1.1 ± 0.4 (11)	1.1 ± 0.4 (12)
Fine sea salt		1.8 (1)	4.6 ± 0.7 (24)	4.5 ± 0.9 (25)
Coarse sea salt			0.96 ± 0.18 (21)	1.0 ± 0.2 (21)
Total sea salt			2.1 ± 0.5 (10)	2.1 ± 0.5 (10)

[a]Mode is listed in the table as fine or coarse rather than $PM_{2.5}$ and $PM_{10-2.5}$ because the variety of sampling and estimation methods used may not have always been based on $PM_{2.5}$ or $PM_{10-2.5}$ sampling methods.

Source: Permission pending, Malm and Hand (2007).

There is a broad range in scattering efficiencies across both regions and species in Table 13-1. Part of this variability is due to the different methods and their varying biases and uncertainties used in each study. Therefore, the true variances in mass scattering efficiencies due to microphysical differences in the particles are likely smaller. Based on their review, Hand and Malm (2007) made a series of recommendations for the dry mass scattering efficiencies for the visible wavelengths listed in Table 13-2.

Table 13-2 Mass scattering efficiency recommendations.

Species	Recommendation (m^2/g)	Comment
$PM_{2.5}$ ammonium sulfate	2.5	2 m^2/g in dry, clean environments 3 m^2/g in more polluted environments
$PM_{2.5}$ ammonium nitrate	2.7	
$PM_{2.5}$ organic matter	3.9	assuming carbon multiplier of 1.8
$PM_{2.5}$ soil	3.3	assuming perfect 2.5 μm cut point ~1 m^2/g for IMPROVE, CSN samplers
$PM_{2.5}$ sea salt	4.5	assuming perfect 2.5 μm cut point 1−1.3 m^2/g for more realistic samplers
Mixed $PM_{10-2.5}$ mass	1	large variability depending on RH, PM composition, PM size distribution
Mixed $PM_{2.5}$ mass	3.6	large variability depending on RH, PM composition, PM size distribution

Source: Permission pending, Hand and Malm (2007).

Mass scattering efficiencies from a number of studies in urban and rural environments were reported since the publication of these recommendations (Cheng et al., 2015; Pandolfi et al., 2014; Tao et al., 2014; Titos et al., 2012; Wang et al., 2012; Malm et al., 2009; Wagner et al., 2009; Andreae et al., 2008; Cheng et al., 2008). Overall, within a given species or mix of PM, there is wide variation in results, with over a factor of 2 or more difference between average results across the studies. However, these values are within the range of the study results reviewed by Hand and Malm (2007). In addition, Malm et al. (2011) showed that the organic mass scattering efficiency in Equation 13-7 is also sensitive to changes in the organic composition.

In addition to mass scattering efficiencies required for all major PM species, a full accounting for light extinction also requires mass absorption efficiencies for species that absorb light. Light absorption by PM is due mostly to black carbon (BC), although some contribution from organic matter is also possible (Petzold et al., 2013). Soil or dust particles in the atmosphere also contribute to potentially substantial amounts of atmospheric absorption (Fialho et al., 2014; Moosmueller et al., 2012). While light absorption by elemental carbon is included as a term in Equation 13-7, several estimates of mass absorption efficiencies for light absorbing carbon (LAC) were published before publication of the 2009 PM ISA, but were not included in the document. To fill this gap, those earlier studies are included for the first time in this ISA along with more recent observations.

Bond and Bergstrom (2006), attempted to understand and reconcile the wide range of reported LAC absorption efficiencies and recommended a mass absorption efficiency of 7.5 ± 1.2 m^2/g for LAC. This recommendation is consistent with results of Andreae et al. (2008), who estimated the LAC absorption efficiency to be 8.5 m^2/g. When organics and LAC were incorporated into a multilinear regression analysis, the LAC absorption efficiency reduced to 7.7 m^2/g. In Fresno, California, Chow et al. (2009) derived a LAC absorption efficiency of 7.9 ± 1.5 m^2/g. The large range of values for light absorbing carbon (LAC) mass absorption efficiencies is due in large part to LAC mass concentration measurements being method dependent, as well as to dependence of the absorption efficiency on wavelength and size distribution.

Absorption is often assumed to be due to particulate black carbon that absorbs in all visible wavelengths. However, there is increasing evidence that organic carbon compounds such as organonitrates absorb light in the near-ultraviolet–blue wavelengths (Lack et al., 2013; Claeys et al., 2012; Kitanovski et al., 2012). This absorption can be significant, with organic mass absorption efficiencies at ~400 nm in a smoke plume varying between 0.25 m^2/g and 2.9 m^2/g (Lack et al., 2013; Yang et al., 2009; Hoffer et al., 2006; Kirchstetter et al., 2004)). It is also missed by measurement methods that focus on green wavelengths, i.e., $\lambda \sim 550$ nm. The absorption of brown carbon in the blue wavelengths is important from a radiation balance standpoint. However, since brown carbon has little absorption in the green and red wavelengths, this should have only a small effect on visibility.

13.2.3.2 Hygroscopic Growth

The relative humidity growth functions in Equation 13-7 are the same for both sulfate and nitrate and are based on experimental growth curves for ammonium sulfate in their most hydrated state (Pitchford et al., 2007; Malm et al., 1994). The growth curves used are supported by a number of recent field studies (Lowenthal et al. (2015); (Chen et al., 2014; Liu et al., 2013; Liu et al., 2012; Stock et al., 2011; Achtert et al., 2009).Numerous laboratory studies have also shown that organic coatings on inorganic particles induce a lower deliquescence point compared to that of the pure inorganic compounds (Li et al., 2014; Peckhaus et al., 2012; Smith et al., 2012; Wu et al., 2011; Pope et al., 2010), and mixed-salt particles generally deliquesce at lower relative humidity than the single-salt particles (Freney et al., 2009). Consequently, outside of very dry environments, even ambient, fully neutralized inorganic salts would generally exhibit smooth growth with relative humidity.

Water uptake by particulate organic matter is not well understood, and in Equation 13-7 the size of organic particles is assumed to be independent of relative humidity, based on the observed the relationship between relative humidity and PM mass with high organic content (Reid et al., 2005; Malm et al., 2003). More recent studies suggest that organic mass is at least slightly hygroscopic, with observations of wet particle diameter/dry particle diameter of water soluble organic PM and humic-like substances from urban, rural, and biomass burning samples ranging from 1.08 to 1.10 at RH of 80%

(Lowenthal et al., 2015; Hallar et al., 2013), 1.13 to 1.19 at RH of 90% (Lowenthal et al., 2015; Kristensen et al., 2012), and 1.25 at RH of 95% (Kristensen et al., 2012) Organics are a significant contributor to urban $PM_{2.5}$ (see Chapter 2) and the exclusion of an f(RH) term for organics in Equation 13-6 likely results in an underestimation of the urban reconstructed b_{ext}.

13.2.3.3 Reconstructing b_{ext} from PM Speciation Data

In addition to the slight modification to develop Equation 13-7 from Equation 13-6, other revisions or rearrangements have been developed as attempts to improve performance or convenience, and results to these changes have been evaluated recently. Equation 13-6 tended to underestimate the highest light scattering values and overestimate the lowest values at IMPROVE monitors throughout the U.S. (Malm and Hand, 2007; Ryan et al., 2005; Lowenthal and Kumar, 2004), in the polluted Pearl River Delta region, and in Shanghai, China using 24-hour $PM_{2.5}$ filter samples (Deng et al., 2013) or $PM_{2.5}$ speciation data from semicontinuous monitors with higher time resolution (Cheng et al., 2015; Zhang et al., 2013b). Limited field studies suggested that particle size distributions and associated mass scattering coefficients may increase with concentrations (Lowenthal and Kumar, 2004; Malm et al., 2003). Although little research has been carried out on urban areas in the U.S., a similar shift of particle size distribution to larger sizes with increasing concentrations in rural and urban settings has been consistently observed in more recent studies in Europe and China (Cheng et al., 2015; Tian et al., 2014; Wang et al., 2014a; Wang et al., 2012; Yang et al., 2012; Calvo et al., 2010; Yue et al., 2009; Baeumer et al., 2008).

To resolve these biases, *a revised IMPROVE equation* was developed (Pitchford et al., 2007) that divides PM components into small and large particle sizes with separate mass scattering efficiencies and hygroscopic growth functions for each size. The revised IMPROVE *equation* was described in detail in the 2009 PM ISA (U.S. EPA, 2009), and it both reduced bias at the lowest and highest scattering values and improved the accuracy of the reconstructed b_{ext}. However, poorer precision was observed with the revised IMPROVE equation compared to the original IMPROVE equation, indicating that the revised equation introduced new random errors. The differences resulting from the two equations in identifying the best and worst haze conditions and the apportionment of the various PM components were small (U.S. EPA, 2009).

Lowenthal and Kumar (2016) recently tested assumptions and evaluated the performance of the revised IMPROVE equation in National Parks and suggested further modifications were needed. They observed that the ration of [OM]/[OC] was closer to 2.1 than the currently used value of 1.8. They also observed that water soluble organic matter absorbs water as a function of RH, which is not accounted for in either the original or revised IMPROVE equations. They further reported that sulfate was not always completely neutralized, as assumed by both the original and the revised IMPROVE equation. Their results suggested that light scattering by sulfate was overestimated and light scattering by organic matter was underestimated by the revised IMPROVE equation. They concluded that the revised IMPROVE equation

did not resolve the biases it was intended to address, and that it should be re-examined (Lowenthal and Kumar, 2016).

Equation 13-6 has also been rearranged for convenient use with hourly measured RH, $PM_{2.5}$, and NO_2, and historical monthly averaged particulate composition (So et al., 2015). Overall, r^2 for all study sites, including those without site-specific speciation data, ranged from 0.72 to 0.77, and absolute normalized mean bias and normalized mean error were generally less than 5% and 25%, respectively, at all sites. Although NO_2 extinction was included in the study, it was mainly used to determine how much of the total extinction was due to $PM_{2.5}$, and conclusions were limited to $PM_{2.5}$ extinction.

In Equation 13-6 and Equation 13-7 it is assumed that the particle species are externally mixed, but this is generally not the case (Degheidy et al., 2015). Although previous studies have indicated that differences among the calculated light extinction values using external and various internal mixture assumptions are generally less than about 10% (U.S. EPA, 2009), newer work suggests potential nonlinearities in the resulting refractive indices of mixed particles. Freedman et al. (2009) found that the refractive indices of internal mixtures of ammonium sulfate and succinic acid were higher than for either pure compound alone at high organic mass fractions and that for mixtures of oxalic or adipic acid with ammonium sulfate, the refractive indices of the mixtures were about the same as ammonium sulfate for all organic mass fractions. Freedman et al. (2009) also calculated that a distribution of mixed particles containing 25% ammonium sulfate and 75% succinic acid resulted in 40% more scattering than would be estimated using volume-weighted, average refractive indices.

13.2.4 Seasonal and Spatial Patterns of Visibility Impairment

In this section light extinction is apportioned to PM species using data from the from the IMPROVE and CSN monitoring networks described in Chapter 2 (Section 2.4). Concentrations for all reconstructed particulate components used for estimating b_{ext} are determined using calculations listed in Table 13-3, which are based on the analyses and procedures laid out in the IMPROVE Report V (Hand et al., 2011) and related publications (Hand et al., 2014b; Hand et al., 2014a; Hand et al., 2013; Hand et al., 2012a; Hand et al., 2012b; Hand et al., 2012c). For example, the mass of ammonium sulfate (AS) is used in Equation 13-7 along with masses of other $PM_{2.5}$ species in the first column of Table 13-3 to estimate light extinction. However, the species actually measured in the CSN and IMPROVE networks is sulfate SO_4^{2-} rather than AS, which is NH_4^+ added to SO_4^{2-} and has a greater mass. Column 2 shows that the concentration of ammonium sulfate [AS] is calculated from the concentration of sulfate [SO_4^{2-}] by multiplying [SO_4^{2-}] by 1.375, which is the ratio of the equivalent mass of [AS] to the equivalent mass of [SO_4^{2-}], i.e., adding ammonium to sulfate increases its mass by a factor of 1.375.

Table 13-3 Composite PM components.

$PM_{2.5}$ Species[a]	Calculation[b]	Assumptions
Ammonium sulfate AS = $(NH_4)_2(SO_4)$	$1.375[SO_4^{2-}]$	Sulfate is assumed to be fully neutralized for both IMPROVE and CSN data.
Ammonium nitrate AN = NH_4NO_3	$1.29[NO_3^-]$	Nitrate is assumed to be ammonium nitrate for both IMPROVE and CSN data.
Particulate organic matter (POM)	1.8[OC]	Derived from organic carbon (OC), assuming an average organic molecule is 55% carbon.
Light absorbing carbon (LAC)	LAC	
Fine particulate soil	2.2[Al] + 2.49[Si] + 1.63[Ca] + 2.42[Fe] + 1.94[Ti]	Fine soil is composed of common metal oxides; FeO and Fe_2O_3 are equally abundant; soil potassium = 0.6[Fe]; a factor of 1.16 is used to account for other compounds such as MgO, Na_2O, CO_3. Same assumption for both IMPROVE and CSN data.
Sea salt (SS)	$1.8[Cl^-]$ or 1.8[Cl]	Sea salt is 55% chloride by weight. IMPROVE sea salt is computed from chloride ion data, while CSN is computed from chlorine concentrations, since Cl^- is not available.
Dry reconstructed fine mass (RCFM)	[AS] + [AN] + [POM] + [LAC] + [Soil] + [SS]	
Coarse mass	$[PM_{10}] - [PM_{2.5}]$	

[a]Species used in Equation 13-7.

[b]The species measured in IMPROVE and CSN network is not exactly the same as the species used in Equation 13-7. The calculation column lists the factor multiplied by the measured species to give the calculated species concentration actually used in Equation 13-7. For example, sulfate is measured in the IMPROVE and CSN networks, but available mass scattering efficiencies are for ammonium sulfate. Therefore, the measured sulfate concentrations must be converted to ammonium sulfate by calculating the corresponding ammonium sulfate mass from the measured sulfate mass.

Sources: Hand et al. (2014b); Hand et al. (2014a); Hand et al. (2013); Hand et al. (2012a); Hand et al. (2012b); Hand et al. (2012c); Hand et al. (2011)

$PM_{2.5}$ mass reconstruction methods were recently reviewed, uncertainties in $PM_{2.5}$ mass concentration, and reconstructed PM components in the IMPROVE and CSN networks using multiple linear regression methods (Chow et al., 2015; Malm et al., 2011). In addition, several field studies in rural environments tested some of the assumptions in Table 13-3, concluding that ammonium sulfate was fully neutralized and particle size with increasing RH followed a smooth growth curve (Lowenthal et al., 2015; Lowenthal et al., 2009). $PM_{2.5}$ concentrations are directly measured in the IMPROVE network. Particulate sulfate is assumed to be fully neutralized ammonium sulfate and estimated from the sulfate ion

measurement. Particulate nitrate is assumed to be in the form of ammonium nitrate from the reaction of nitric acid and ammonia gas. Organic mass is estimated by scaling the OC from the thermal optical reflectance analysis to particulate organic mass (POM) where the scale factor accounts for oxygen, hydrogen, and other noncarbon molecules. It was assumed that the ratio of POM divided by OC mass (ROC) was 1.8, or 55% of POM was carbon. This value was based on a regression analysis of the major PM composite components against measured $PM_{2.5}$ concentrations in the IMPROVE network (Malm and Hand, 2007).

LAC is the EC concentration reported from the thermal optical analysis of organic carbon (OC) and elemental carbon (EC) (Watson et al., 2005). Soil mass concentrations are estimated by a general method that sums the oxides of elements that are typically associated with soil (Al_2O_3, SiO_2, CaO, K_2O, FeO, Fe_2O_3, TiO_2). To account for other compounds such as MgO, Na_2O, and carbonates, the sum is multiplied by a factor of 1.16 (Malm et al., 1994). Molar concentrations of iron are assumed to be equally abundant in the forms of FeO and Fe_2O_3, and soil potassium is estimated by using Fe as a surrogate, or [K] = 0.6[Fe], because unlike Fe and other soil elements, the K in $PM_{2.5}$ is also contributed in abundance by another source, biomass burning (Malm et al., 1994). Sea salt concentrations are typically computed from sea salt markers, with the most common being sodium (Na). The Na ion is not routinely measured in the IMPROVE program, and elemental Na is poorly detected by IMPROVE's routine X-ray fluorescence analysis (White, 2008), so the chloride ion is used instead (Table 13-3).

The chloride ion has been shown to be a good predictor of conserved sea salt mass near coastal areas (White, 2008) but can be lost during atmospheric aging due to reactions with nitric acid, which produces particulate sodium nitrate and gaseous hydrochloric acid. The use of the chloride ion likely results in an underestimation of sea salt's contribution to $PM_{2.5}$ farther away from coastal areas, but sea salt concentrations are generally reduced by dispersion and removal processes, leading to smaller contributions to $PM_{2.5}$. Elemental chlorine concentrations are used to estimate sea salt for CSN data, because the chloride ion is not analyzed by the CSN. Comparisons of sea salt concentrations between 25 collocated CSN and IMPROVE sites located throughout the U.S. observed that IMPROVE concentrations were up to three times higher on average compared to CSN, with a relative bias of 63%, or large enough for the data to be considered semiquantitative (Hand et al., 2011). Difficulties in measuring sea salt in the IMPROVE and CSN networks including the lack of Na^+ measurements as a check and depletion of Cl^- due to displacement by NO_3^- are discussed by Hand et al. (2011).

13.2.4.1 Seasonal and Spatial Light Extinction $PM_{2.5}$ Species Contributions

Approximately every five years the IMPROVE program releases a report summarizing the spatial and temporal patterns of $PM_{2.5}$ composition and its contribution to light extinction from IMPROVE and CSN monitoring sites, which are mostly urban and rural, respectively. The latest report, IMPROVE Report V, was published in 2011 (Hand et al., 2011) and included a summary of the seasonal and

geographic distributions of species contributions to $PM_{2.5}$ and light extinction for IMPROVE and CSN monitoring sites averaged over the years 2005–2008. The b_{ext} associated with $PM_{2.5}$ components was calculated using Equation 13-7 and the same monthly climatological f(RH) curves used in the Regional Haze Rule guidance document (U.S. EPA, 2003). These data can be used to identify differences between urban and rural light extinction species contributions by region and season. This contrasts with most visibility data, including data presented in the 2009 PM ISA (U.S. EPA, 2009), which have historically been based mainly on rural and remote measurements.

Twenty-eight IMPROVE regions were empirically defined based on-site location and magnitudes and seasonal distribution of PM concentrations for major species. Elevation was not explicitly taken into account in these groupings. Thirty-one CSN regions were defined based on seasonal distributions of PM concentrations and site locations. For comparison purposes and where possible, CSN regions were defined similarly to those for the IMPROVE network (Hand et al., 2011). Although the ability to leverage the sampling networks to provide extinction estimates provides valuable insight, these mass-based estimates are less accurate than calculations that use particle size and composition information.

Hand et al. (2012c) published the finding for the seasonal $PM_{2.5}$ species concentrations for the IMPROVE and CSN regions using PM species listed in Table 13-3, averaged over the years 2005−2008. The data were aggregated over regions or groupings of IMPROVE or CSN monitoring sites. Twenty-eight IMPROVE regions were empirically defined based on-site location and magnitudes and seasonal distribution of aerosol concentrations for major species. Elevation was not explicitly taken into account in these groupings. Thirty-one CSN regions were defined based on seasonal distributions of aerosol concentrations and site locations. Of the thirty-one CSN regions, eight had only one site per region because seasonal distributions were unique in comparison to the nearest other monitors, and these regions are identified by individual cities. Where possible, CSN regions were defined similarly to those for the IMPROVE network for comparison purposes.

Following is a summary of the $PM_{2.5}$ b_{ext} species contribution estimates from Hand et al. (2011). The b_{ext} species contributions differ from the $PM_{2.5}$ mass contributions in that the relative contribution of fine soil scattering is reduced due to its comparatively low scattering efficiency, and the relative contributions of ammonium sulfate and nitrate are increased due to the f(RH) factors. The results are presented as monthly stacked bar charts for each region in Figure 13-1, Figure 13-2, Figure 13-3, Figure 13-4, Figure 13-5, Figure 13-6, Figure 13-7, Figure 13-8, Figure 13-9, Figure 13-10, Figure 13-11, and Figure 13-12. The figures are arranged in pairs, with odd-numbered figures showing data for 2011−2014 and even-numbered figures for the same region and monitors showing data for 2005−2008. The most recent data are presented first because the discussion focuses mainly on the 2011−2014 data shown in the odd-numbered figures, but earlier data for 2005−2008 are shown for comparison. Figure 13-1, Figure 13-2, Figure 13-3, Figure 13-4, Figure 13-5, and Figure 13-6 summarize the IMPROVE b_{ext} species contributions, while Figure 13-7, Figure 13-8, Figure 13-9, Figure 13-10, Figure 13-11, Figure 13-12, Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean.

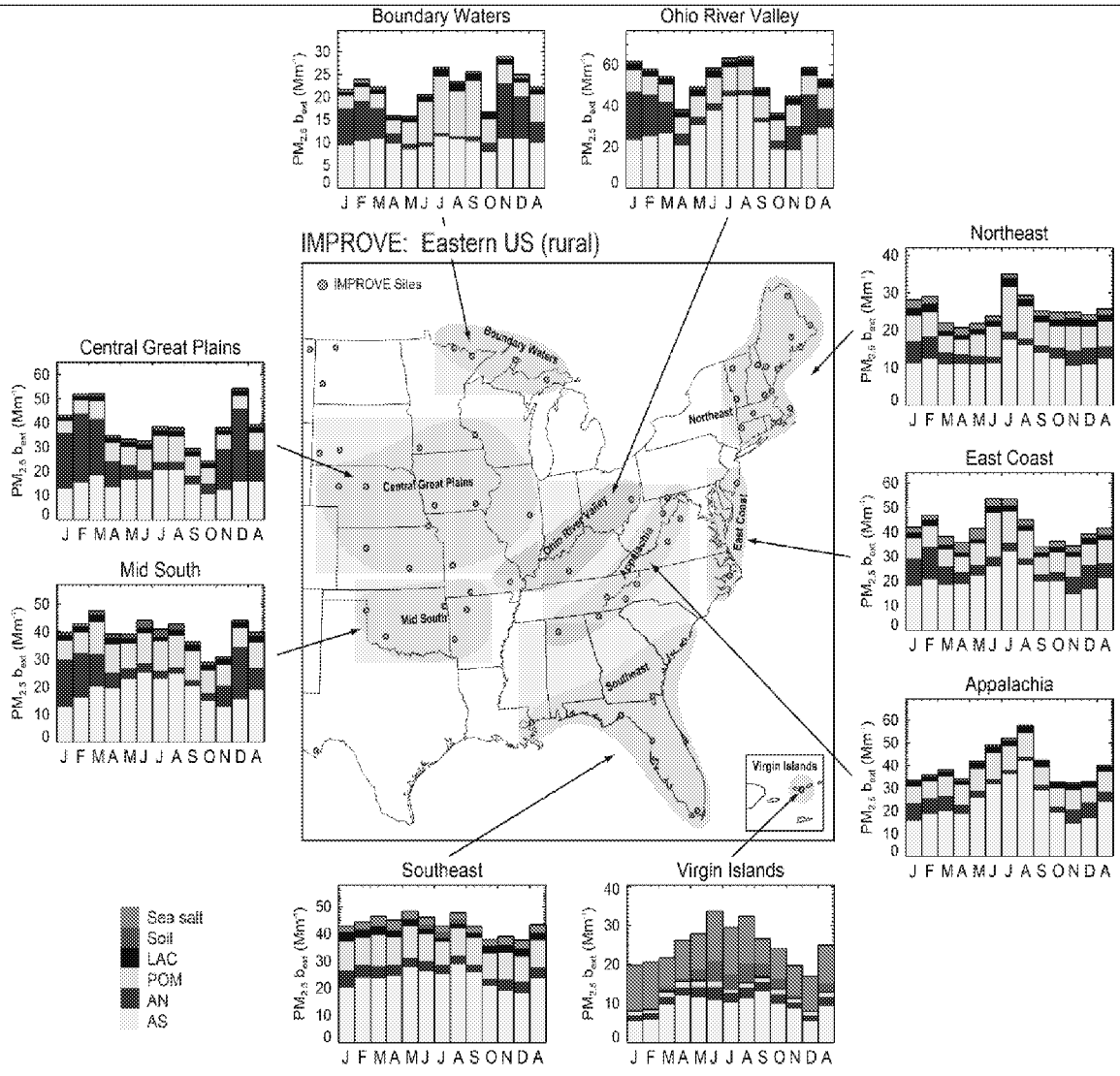
Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Hand et al. (2011).

Figure 13-13, and Figure 13-14 summarize the CSN b_{ext} species contributions. Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Hand et al. (2011).

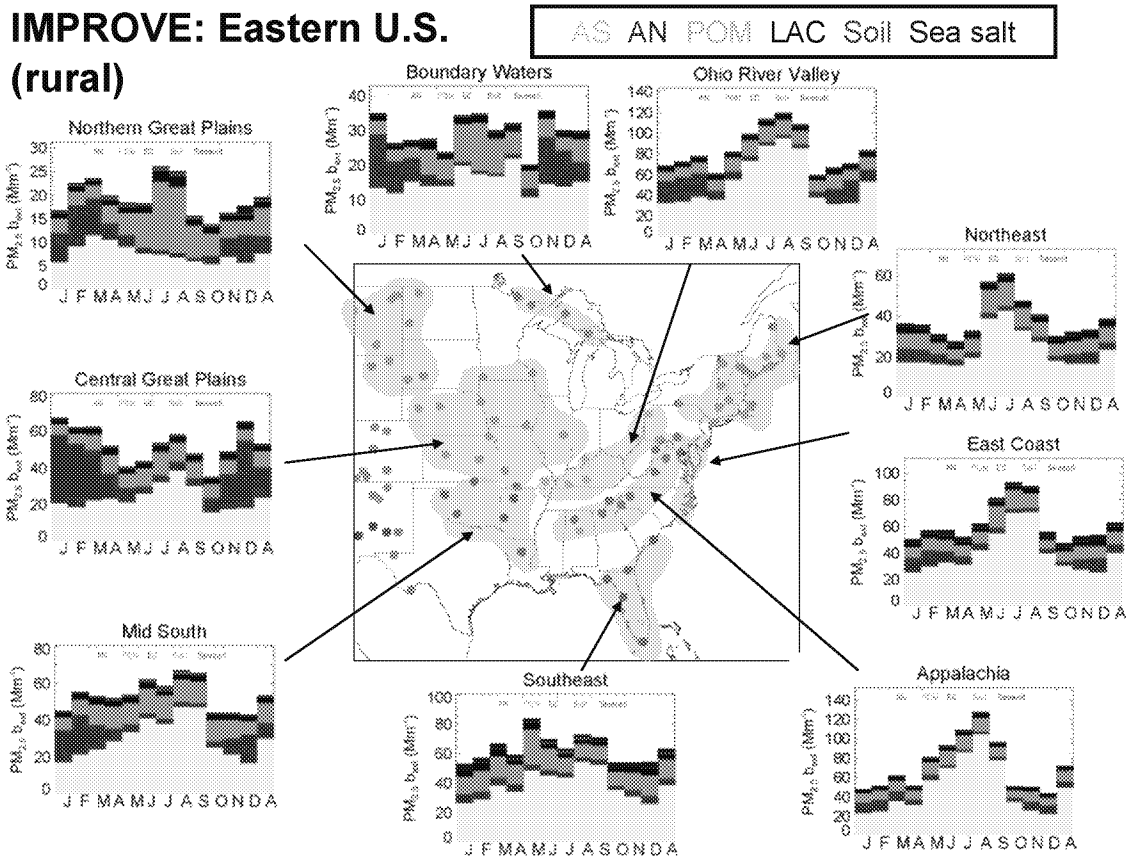
Figure 13-13 and Figure 13-14 show b_{ext} budgets for Alaska, Hawaii, and the Virgin Islands for 2005–2008 from the IMPROVE and CSN networks, respectively. These were presented separately in the original publication by Hand et al. (2011), but are included if available with other regions in the updated figures from 2011–2014 (Figure 13-3, Figure 13-5, and Figure 13-9).



Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Update of Hand et al. (2011).

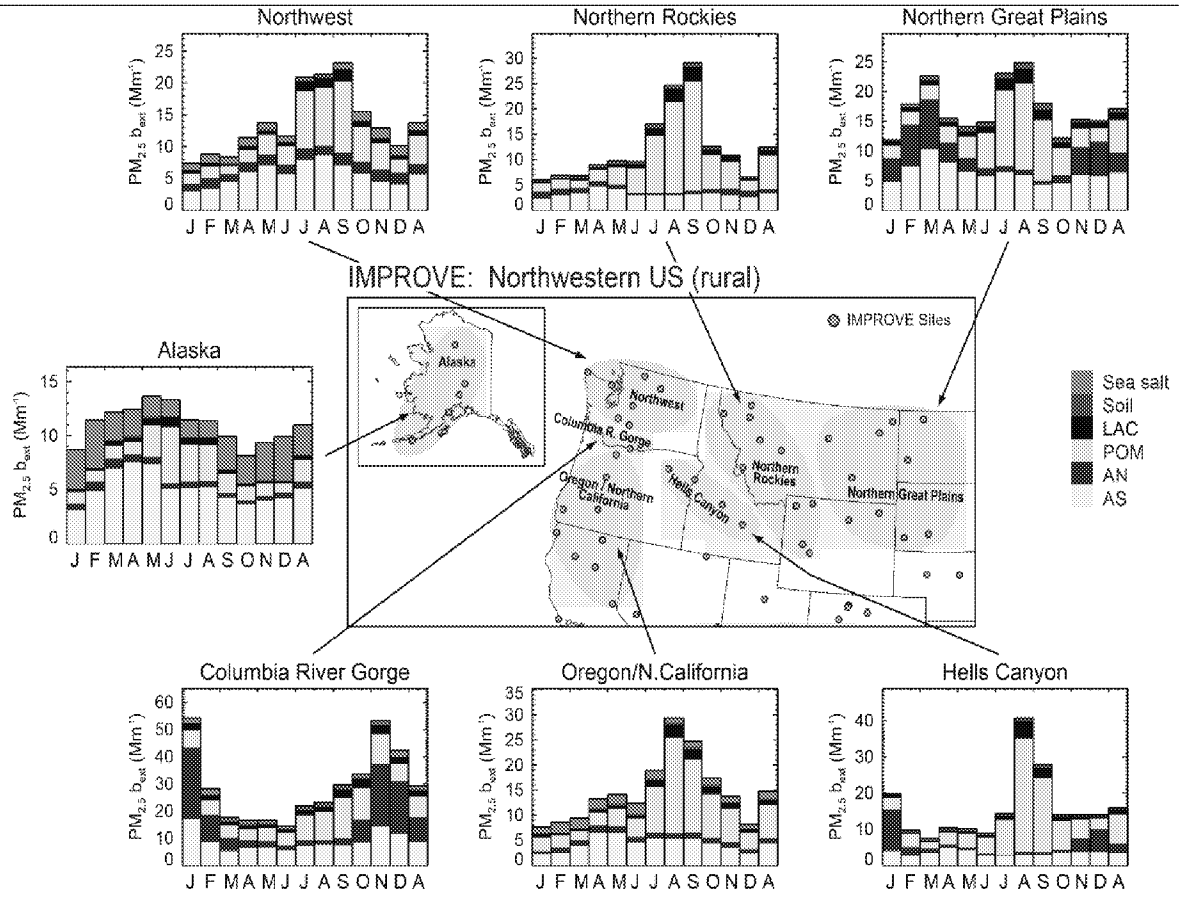
Figure 13-1 IMPROVE 2011–2014 regional monthly mean $PM_{2.5}$ reconstructed light extinction coefficients (b_{ext}, Mm^{-1}) for the Eastern U.S.



Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending Hand et al. (2011).

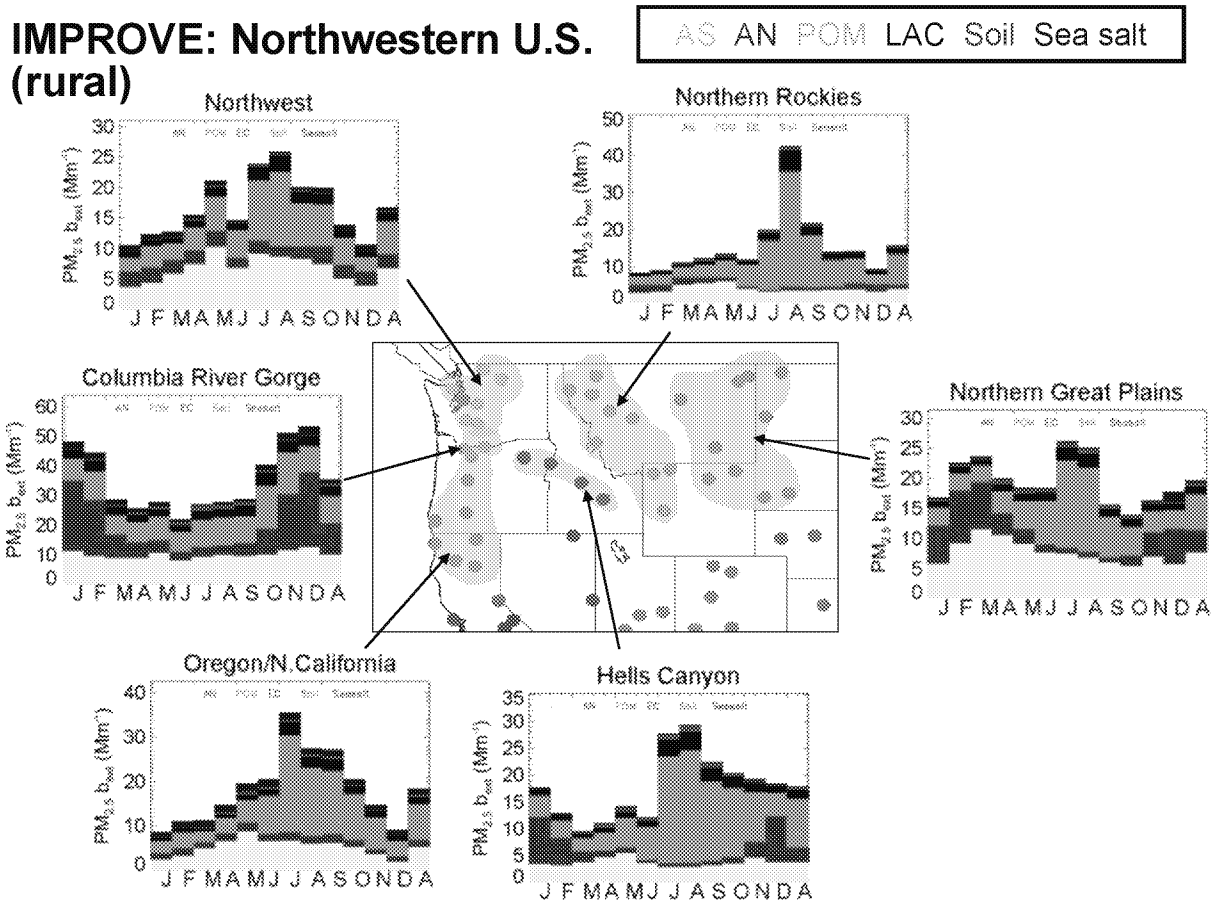
Figure 13-2 IMPROVE 2005–2008 regional monthly mean $PM_{2.5}$ reconstructed light extinction coefficients (b_{ext}, Mm^{-1}) for the Eastern U.S.



Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Update of Hand et al. (2011).

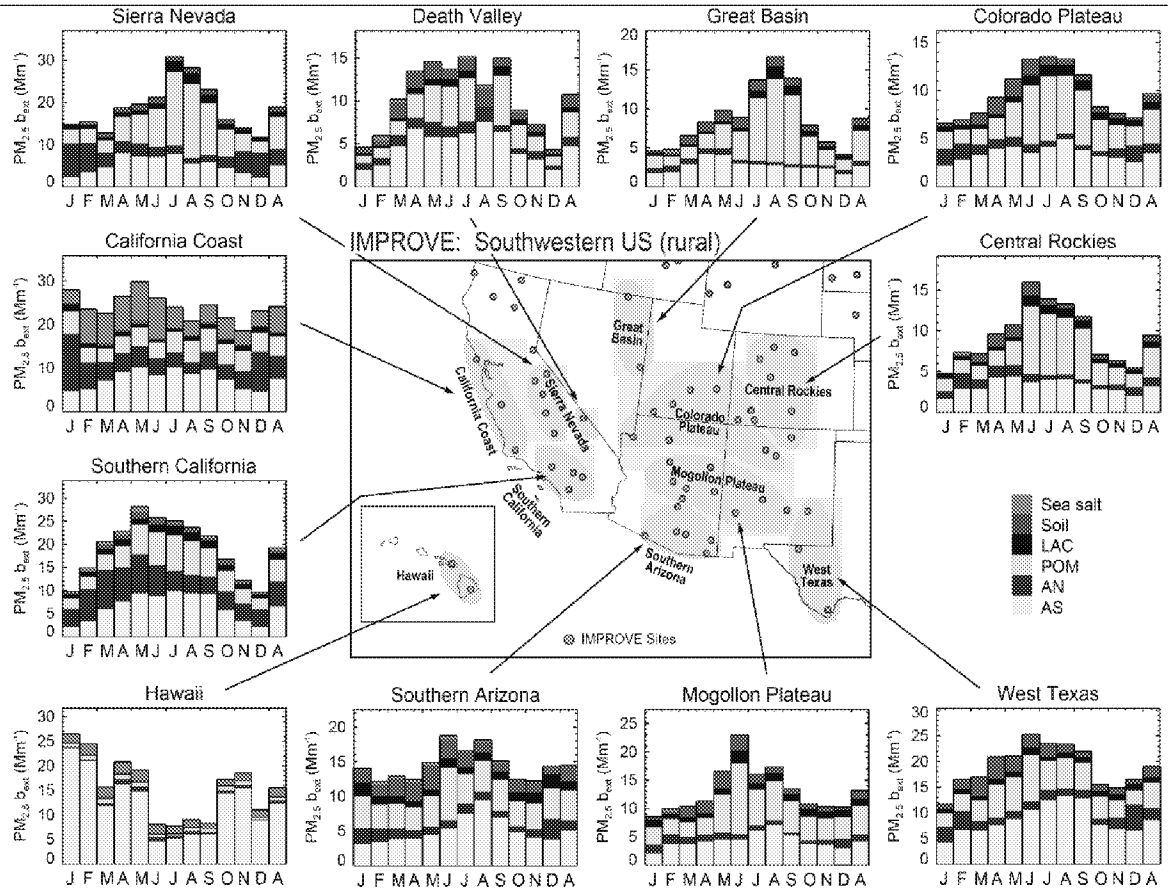
Figure 13-3 IMPROVE 2011–2014 regional monthly mean $PM_{2.5}$ reconstructed light extinction coefficients (b_{ext}, Mm^{-1}) for the Northwestern U.S.



Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Hand et al. (2011).

Figure 13-4 IMPROVE 2005–2008 regional monthly mean $PM_{2.5}$ reconstructed light extinction coefficients (b_{ext}, Mm^{-1}) for the Northwestern U.S.



Note: The letters on the x-axis correspond to the month and "A" corresponds to "annual" mean. Ammonium sulfate (AS) in yellow, ammonium nitrate (AN) in red, particulate organic matter (POM) in green, light absorbing carbon (LAC) in black, soil in brown, and sea salt in blue. The shaded area corresponds to the regions that comprise the sites used in the analysis, shown as dots. Extinction coefficients were determined from Equation 13-7 (see text). Wavelength corresponds to 550 nm.

Source: Permission pending, Update of Hand et al. (2011).

Figure 13-5 IMPROVE 2011–2014 regional monthly mean $PM_{2.5}$ reconstructed light extinction coefficients (b_{ext}, Mm^{-1}) for the Northwestern U.S.